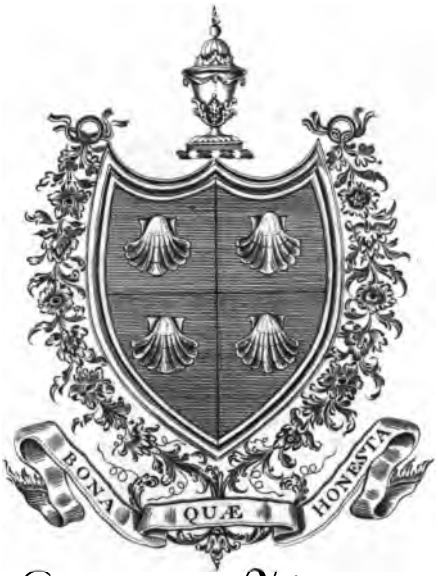
CLINICAL DISORDERS OF THE HEART BEAT.

THOMAS LEWIS.



Henry Jackson

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Nov. 1912.

CLINICAL DISORDERS

OF THE

HEART BEAT.

A HANDBOOK FOR PRACTITIONERS AND STUDENTS.

BY

THOMAS LEWIS, M.D., D.Sc., M.R.C.P.

Lecturer in Cardiac Pathology, University College Hospital Medical School,
Physician to Out-Patients, City of London Hospital
for Diseases of the Chest.

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PREFACE.

THERE can be but few, engaged in the active practice of medicine, who are not aware that a new and important chapter has been added to our knowledge of the mechanism of the heart beat during recent years. The newly-acquired information has been gathered by the employment of precise graphic methods. Those, who are engaged in studying the heart and its defects by means of special instruments, are fully conscious of the burden which awaits the student or practitioner who has yet to bring himself abreast of the times in this field of knowledge.

A question is often put to some of us. In what degree is an acquaintanceship with the new methods essential or expedient in the routine of busy practice? The graphic study of heart affections is but one of many clinical and pathological subjects which has

forged ahead of late years. While a medical man can ill afford to neglect the advance of a subject in which he practises, he may act, in a too vigorous pursuit of one branch of medical science, to the detriment of his knowledge in other directions. A universal and detailed acquaintanceship with medical science as it exists to-day is no longer possible, but it behoves all practitioners to grasp new principles and to be aware of their influence upon the care of patients afflicted with common maladies.

If I am asked whether it is essential that a practitioner of general medicine should be trained to record the movements of the several heart chambers, I am inclined to reply that the acquisition of the special manipulative skill and the necessary experience, which the obtaining and accurate interpretation of graphic records involves, entails too great an expenditure of time and energy adequately to repay him or the patients he serves. And my reply is dictated by the belief that most of those disturbances of the heart's mechanism which are met with in every-day practice can be identified by simpler means.

Reflections of this kind influence me in offering to medical men a small handbook, which I trust may inform them of the new facts and conclusions which are of chief service at the bedside.

I have confined the reproduction of graphic records almost to such as illustrate what may be seen and felt, for many disorders of the heart can be identified by sight and touch when these senses are aided by hearing. A single and portable piece of apparatus may be used in cases of doubt and difficulty, to supplement the observations so obtained. The Dudgeon sphygmograph is probably familiar to most medical men; fitted with elastic bands of attachment, and preferably with a time-marker also*, it readily allows a short strip of radial pulse curve to be obtained. Such a curve, alone, will usually place the observer in possession of facts, which are sufficient for an analysis of the common disturbances of the heart beat. The use of the sphygmograph encourages

* The complete apparatus is supplied by S. Shaw, Esq., of Padiham, Lancashire, at a moderate cost.

accuracy; yet, as I hope to show, a great deal can be accomplished without it.

In the succeeding chapters, I have not attempted to acquaint the reader with the evidence* upon which the diagnosis of the several cardiac disorders rests, but have recounted such physical signs as I have found serviceable in identifying these disorders, prior to the application of more precise methods in individual instances.

To ascertain the nature of the heart's mechanism in a given patient is, as we shall see, of twofold value. It is of importance in elucidating the remaining physical signs, especially those of auscultation. And it seriously affects the attitude towards the patient, for it often influences the prognosis and treatment profoundly. The recognition of the existing mechanism is, as experience has so often shown, one of the very first essentials in the care of cardiac cases.

Such new facts, as may be found in the chapters, have been collected largely during the tenure of a Beit

* A full account of such evidence may be found by those who desire it in my recent publication, "The Mechanism of the Heart Beat," published by Messrs. Shaw and Sons, London.

Memorial Fellowship, to the Trustees of which it is my desire to state this obligation. That the reader may benefit from a fuller experience, I have not hesitated to take advantage of the published works of other writers on the same subjects. It is pleasurable to acknowledge the kindness of my friend and colleague Dr. T. R. Elliott, for his criticism of the chapters and for his careful perusal of the proof sheets; and I am grateful to all my colleagues at University College Hospital for the generous manner in which they have placed their material at my disposal.

TH. LEWIS.

58*a* *Wimpole Street,*
December, 1911.

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CHAPTER I.

THE RECOGNITION AND IDENTIFICATION OF DISORDERS OF THE CARDIAC MECHANISM.

It seems desirable that I should open the chapters of this book, by acquainting my readers with the general arrangement of the matter contained in it.

Since those who work amongst the sick usually discover a real interest in a particular phenomenon by observing it rather than by reading of it, I begin the first chapter with a general description of the chief derangements of the rate and sequence of the pulse and heart beat as they are felt, seen, or heard by all practitioners. I take certain common and generally recognised physical signs as they are noted at the bedside and translate these signs into terms of mechanism, without attempting to describe the manner in which they are brought about and without suggesting their value in prognosis or treatment. I shall speak of six forms of cardiac disorder, and they will be described under the following headings :—

1. *Sinus arrhythmia.*
2. *Heart-block.*
3. *Premature contractions.*
4. *Paroxysmal tachycardia.*
5. *Auricular fibrillation.*
6. *Alternation of the pulse.*

There may be some to whom these terms are still unfamiliar or to whom their meaning is still obscure. My

immediate endeavour, therefore, is to offer them a preliminary idea of the meaning of these names, an idea which I hope may appeal to them from their past experience; and I do so by citing clear examples of phenomena to which in future I must perforce refer by the use of distinctive names.

If we feel the pulse of a young adult, when his inspiration is deep, or, better still, if we feel the beating of a dog's heart against its chest wall, a periodic irregularity of the pulsations is observed which follows the separate acts of breathing. I cite this disorder of the heart beat as a characteristic example of *sinus arrhythmia*, or one in which the whole heart is involved.

In many patients in whom the radial pulsations and heart beats run with perfect uniformity for long periods, an occasional and isolated disturbance is noticed. The *pulse* intermits; it loses one of its serial beats at intervals. When the *heart* is examined it is found that at the moment of the disturbance a ventricular contraction appears before the rhythmic beat is due, and that this early beat is followed by a pause of unusual length. I cite this disturbance as a simple example of what I shall in future describe as a *premature contraction*, and what has usually been referred to in the past as an "extrasystole."

If in a similar case, where the occasional pulse fails, a similar failure of ventricular action is discovered, so that on listening at the apex beat no abnormal sounds are heard, but the heart remains silent throughout the whole of the pause, the phenomena are evidences of another condition, namely *heart-block*. But lest I should create confusion at an early stage, I must add to this example the statement that heart-block manifests itself in many other ways and in no way which stands in more open contrast to the example now cited than by its production of regular pulses of conspicuously slow rate.

Paroxysmal tachycardia is a term which is probably familiar to all, but I employ it in a restricted sense, and speak only of instances in which an absolutely abrupt acceleration of regular heart beat, which subsequently terminates in an equally abrupt manner, is repeated from time to time.

When a patient, who is known to have mitral stenosis and who requires treatment for cardiac failure, exhibits not only dropsy, venous engorgement and cyanosis, but a rapid and utterly disordered heart action in which there is no rhythmic sequence, he presents the characteristic picture of *auricular fibrillation.*

Finally, if in a case of renal disease or arterio-sclerosis, the pulse tension is high, Cheyne-Stokes' breathing perhaps is present, and the pulse is regular in rhythm, but varies in *force* so that each alternate beat is strong and each alternate beat is relatively weak, an example of *alternation of the pulse* is under observation.

I have deliberately chosen these examples because they are distinctive ; but the several forms of disturbance are not always so clearly differentiated. Were it so, my task would be simple. The examples are distinctive and consequently allow a preliminary idea of the meaning of my terms to be entertained. It is into these terms that I shall in the first instance translate the commoner physical signs, and I do so with the object of providing the student or practitioner with an immediate clue to the type of mechanism with which he is dealing. But as the preliminary description will be inadequate, it is supplemented by a detailed discussion of each form of disorder in the remaining chapters of the book, in which an account of the pathology, prognosis and management will be found.

Preliminary evidences.

Age and frequency. The first guides to the identification of a disordered heart mechanism are the age of the patient in whom it occurs and a knowledge of the frequency of irregularities at different ages.

An irregularity of heart or pulse found before the tenth year is almost always a sinus arrhythmia. Heart-block may be present during the first decade but it is rare; a few premature contractions have been noted in quite young children, in most of whom extensive enlargement of the heart has been present. The earliest age at which auricular fibrillation has been seen is 13 and it is very rare before the age of 17.

The relative frequency of disorders of the cardiac mechanism from adolescence to old age is in general hospital practice approximately as follows :—

Auricular fibrillation	41%
Premature contractions	34%
Paroxysmal tachycardia	10%
Sinus arrhythmia, heart-block and alternation, each	5%

Dealing with those in whom there is obvious evidence of cardiac failure, at least 60% of irregular hearts are irregular because the auricles are fibrillating.

Heart rate. The second clue is the rate of the heart beat. *When the ventricle beats regularly* and its rate is continually below 35 beats per minute, complete heart-block (see Chapter III) is probably present; under similar circumstances a rate which lies between 40 and 50 should arouse a suspicion of partial heart-block; a persistent rate of 130 and over should always bring to mind the possibility that a long continued paroxysm of tachycardia is present.

If, on the other hand, *the ventricle* beats irregularly*, and the rate surpasses 120 per minute, fibrillation of the auricle is probably present, and is the rate is faster, so the probability that such is the mechanism approaches certainty. Irregular hearts, beating at 140 and over, are scarcely ever affected in any other manner. Premature contractions very rarely accompany ventricular rates of 120 and over; sinus arrhythmias are almost confined to rates below 100; and both these forms of irregularity become more frequent as the scale of rate is descended to the sixties and fifties. If an irregularity is observed, and the rate of heart beat is in the neighbourhood of 100, any influences, such as exercise, fever, or the administration of belladonna, which enhance the ventricular rate, tend to abolish all irregularities, with the exception of that due to auricular fibrillation, and in this instance the disorder persists and is often augmented.

Persistence of irregularity. Auricular fibrillation is usually a persistent condition and examination from hour to hour or from day to day reveals its continual presence. The other irregularities are usually transient, the pulse being found to be quite regular from time to time; shorter or longer periods of normal heart action intervene between periods of disturbance.

Common types of disorder and their meaning.

Solitary pulse intermittences. An occasional pause of pronounced length, which interrupts an otherwise perfectly regular pulse, is due to one of two causes,† namely, a premature contraction (common), or a dropped beat as a result of heart-block (rare). They are easily distinguished; the

* I draw especially attention to the distinction between ventricular irregularity and pulse irregularity; they do not always run hand in hand.

† A long expiratory pause of a respiratory arrhythmia may be mistaken for intermittence if the examination is cursory.

premature beat is felt or heard at the apex; it gives rise to an early first, or first and second sound. In block, the heart is silent and motionless during the whole pause.

Coupled beats. If the *ventricular* beats are coupled and the couples are evenly spaced* they are the result of one of two mechanisms, for either the alternate beats of the normal rhythm have been replaced by premature contractions—in which case the second beat of the couple is weak and may not reach the wrist,—or else each third ventricular contraction has been lost and heart-block is present. If the *pulse* beats are coupled (*pulsus bigeminus*) a third possibility remains; the pairing may be due to the occurrence of premature heart beats which replace each third rhythmic beat. If such is the case the premature beat will be appreciable at the apex, though it does not reach the wrist.

Triple beating. The recognition of the cause proceeds along similar lines. Tripling at the apex is due to premature contractions which replace each third rhythmic beat, or to heart-block in which each fourth ventricular contraction has been lost. Tripling at the pulse (*pulsus trigeminus*) may be due to a third cause, namely, premature beats replacing each fourth rhythmic beat, the early beat failing to reach the wrist.

Halved pulse rate. When the ventricle beats at twice the pulse rate, the disorder is due to premature contractions in all but the rarest instances. Alternation has been known to occasion halving, the weak alternate beats failing to reach the wrist; but this condition is of great rarety and so far as I know is only very transient. The two are readily differentiated, for in the first instance the ventricular beats are coupled while in the last they appear regularly.

* Sometimes the pauses following the pairs are not of uniform length. The irregularity is then a complex one; it is due to auricular fibrillation, to which premature beats have been added. When it occurs the patient is usually under the influence of excessive doses of digitalis.

When sudden and exact halving of pulse rate is noted and the ventricular rate is halved simultaneously, the disorder is the result of heart-block.

A grossly irregular pulse in which there is hopeless jumbling of strong pulsations with quick runs of almost imperceptible beats, and in which the lengths of intervening pauses are constantly varying, is due to auricular fibrillation.

A mild grade of irregularity which persists, which is not related to respiration even when the breathing is deepened and in which no definite sequence of events can be determined, is also due to auricular fibrillation in most cases. A similar irregularity, which shows relations to respiration, is a sinus arrhythmia.

In the preceding paragraphs the method of procedure at the bedside is briefly stated, and an acquaintance with the few rules which I have given will enable the practitioner to identify a very large number of the disorderly forms of heart action with which he meets. But where the reader is in doubt, or when he requires more explicit information, in regard to either the arrangements of the beats or the manner of their production, or in respect of the management of the case in which the disorder is discovered, he may refer to the more detailed descriptions contained in the remaining chapters.

CHAPTER II.

SINUS IRREGULARITIES.

Definition.

Irregularities of the heart which are produced by interferences with the rhythmic impulses at the seat of their discharge.

The nature of sinus disturbances.

In a discussion of sinus irregularities, the nerve supply of the heart in relation to disturbances of rhythm occupies a prominent position. Let me state emphatically at the outset that we have nothing to do, first, with the functions of the intrinsic cardiac ganglia, nor secondly with the sympathetic nerve trunks; as we have little or no real knowledge of the part they play in disease, so any theory which ascribes a derangement of the heart to a perversion of their functions is without practical significance. We have a limited but real knowledge of the vagus and its relation to pathology; my remarks upon the cardiac nerves are consequently confined to it.

The complete beat of the normal heart consists of a contraction of its chambers in an orderly sequence. The wave of contraction starts in a small and newly discovered mass of tissue, the *sino-auricular node* (Fig. 1), which lies embedded in the upper and anterior end of the *sulcus terminalis*. The *sulcus terminalis* runs, as may be remembered,

from the junction of the right auricular appendix and the superior vena cava towards the inferior vena cava (see Fig. 1). The tissue of the node, consisting of a specialised network of muscle cells, richly supplied by the nerves of the heart which enter in this region, lies therefore at the mouth of the superior

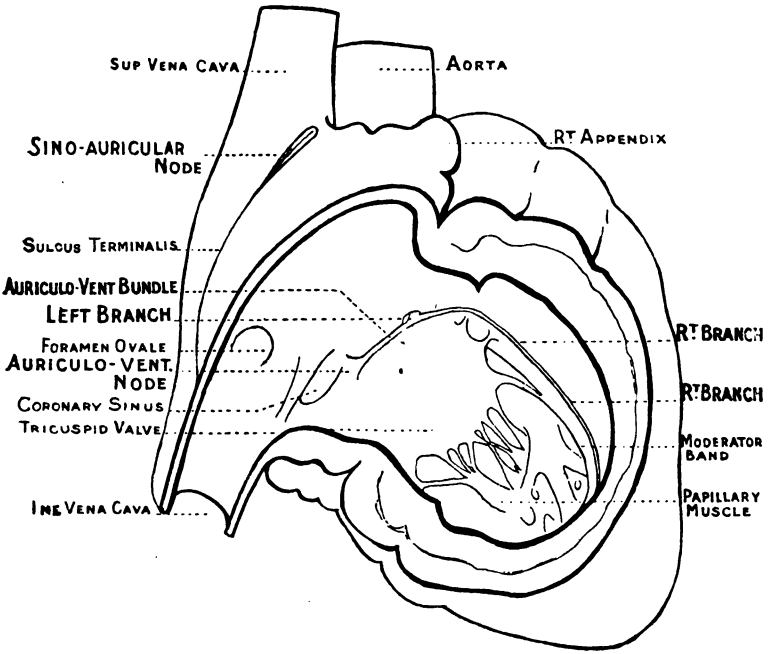

Fig. 1. A diagram of a human heart (modified from Koch's). The walls of inferior vena cava, right auricle and right ventricle have been partially removed to expose the septa. The position of the sino-auricular node, in which the heart beat commences, is shown, as are also the position of the auriculo-ventricular node and the course of the auriculo-ventricular trunk and its branches. The last-named structures convey the contraction wave from auricle to ventricle.

vena cava and is imbedded in the right auricle. The contraction which commences in its neighbourhood spreads through the walls of both auricles and is transmitted by a special band of tissue, which will receive subsequent description, to the ventricles. The orderly rhythm of the whole heart takes its origin in this node, which has consequently been

called the heart's "pacemaker." In the normally acting adult heart, the pacemaker sends forth waves of contraction at rates which average 72 per minute, and, the separate beats being evenly spaced, the systoles follow each other in a regular order or rhythm. The pacemaker is under the control of the vagi, or inhibitory nerves of the heart, and they normally exert a considerable restraining influence upon this stimulus producing centre. Destruction of the nerves, more especially that of the right side, or the administration of atropine, which paralyses the nerve endings in the heart, raises the rate at which the heart beats follow each other. In the human subject, the probable limit to which the rate may rise as a result of this denervation is 150-160 per minute.

In many subjects, and under special conditions, the vagus acts with excessive inhibition, either persistently, or rhythmically. Its influence consequently results either in a uniform pulse slowing or in a waxing and waning of heart rate. Let us deal with uniform slowing first, for it is a subject with which we are only briefly concerned in these lectures. Pronounced slowing of the whole heart is comparatively rare; the lesser grades of slowing, most of which are probably of vagal origin, slowing to 50 or 60 beats per minute, are not uncommon and are especially prominent in association with increased arterial pressure, pregnancy, jaundice, aortic stenosis, convalescence from the acute fevers and less frequently with other conditions. Pulse slowing of this degree has no great significance, and it is not uncommon to meet with people who enjoy perfect health and in whom the pulse rate lies habitually between these limits.

Periodic or varying disturbances, which influence the rhythm of the heart at its source and produce a greater or lesser degree of arrhythmia, are of greater importance, though it will only be necessary to describe the commoner forms of such irregularities.

In Fig. 2, a diagram of a characteristic sinus arrhythmia is given. I shall refer to similarly constructed diagrams in succeeding chapters. The figure is arranged so that each narrow black rectangle (A) represents a single co-ordinate beat of the auricle, and so that each broad black rectangle (V) represents a co-ordinate ventricular contraction. Where an auricular contraction is followed by a ventricular response,

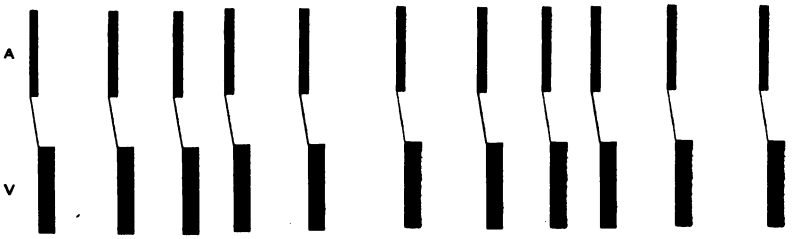

Fig. 2. A diagrammatic representation of the action of a heart, affected by sinus arrhythmia. The contractions of auricle and ventricle are represented by the thin and broader black rectangles, *A* and *V* respectively. The auricle contracts at the beginning of each cycle, and sends its impulse, indicated by an oblique line, to the ventricle, which responds. The irregularity consists of a waxing and waning of rate, in which both auricular and ventricle participate.

an oblique line is drawn, joining the corresponding rectangles. The slope of the oblique line indicates the time interval between the contractions of auricle and ventricle. All such diagrams read from left to right. In the present example, a sinus irregularity, the whole heart is affected, so that each ventricular contraction is preceded by an auricular systole at the usual interval. The irregularity consists of a gradual waxing and waning of auricular rate which is repeated periodically, and which is imitated exactly by the ventricle.

Respiratory irregularities.

It is well known that young adults manifest a very appreciable irregularity of the heart and pulse rhythms when they breathe deeply (Fig. 3). The pulse quickens while the chest is expanded, and slows when the chest is emptied. But in both young adults and old there is no

respiratory variation of pulse rate, which the finger can discover, while *the breathing is natural.* On the contrary, a perceptible degree of natural respiratory irregularity of the pulse, characterised chiefly by one or more long pauses during the expiratory period (Fig. 4 and 5), is not uncommon in young children, and sometimes it is sufficiently prominent to

Fig. 3. A sphygmographic curve from a normal subject, breathing deeply. There is an increase of pulse rate during inspiration and a decrease during expiration.

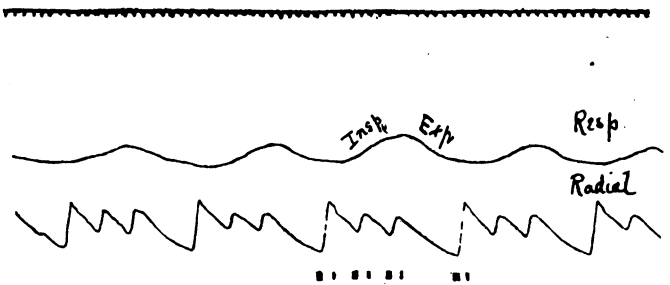

Fig. 6. A gross sinus arrhythmia; a long pulse pause accompanies each expiration. In this, as in all similar figures, the top line represents time in fifths of seconds.

attract immediate attention. Irregularity of a very similar kind is found frequently at the age of puberty, and it is also seen on rare occasions in the adult (a striking example of the last is shown in Fig. 6).

All these irregularities are of vagal origin.

Fig. 4. A curve of an irregularity of the pulse which recurs with each complete cycle of normal respiration.

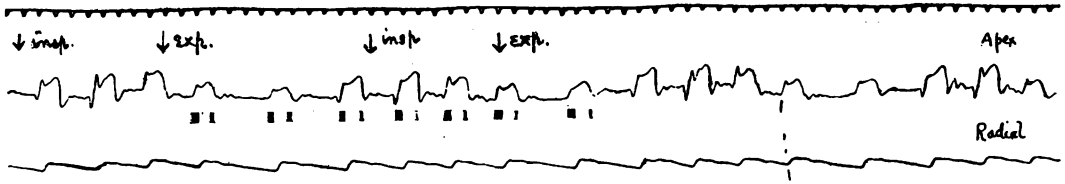

Fig. 5. Curves from the heart's apex beat and radial artery, showing a periodic increase and decrease of pulse rate with the respiratory cycles. The beginnings of inspiration and expiration are indicated by arrows. The heart sounds, which are represented diagrammatically, run parallel with the ventricular beats.

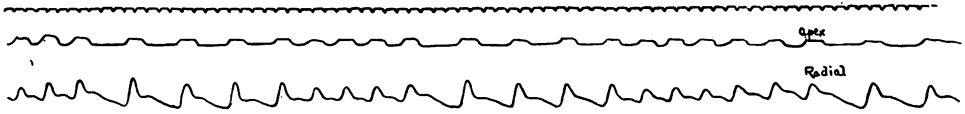

Fig. 7. A periodic irregularity of the heart of sinus origin, which shows no relation to respiration. The breathing was held.

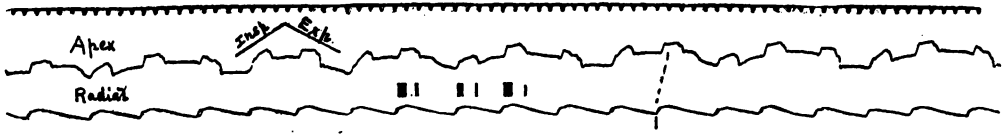

Fig. 8. Slight irregularity of the pulse in a child. The irregularity bore no constant relation to the acts of breathing while it was natural, but became purely respiratory when breathing was deepened. It is a sinus irregularity.

Sinus irregularities which bear no relation to respiration.

While the vagal irregularities of heart rhythm generally show a respiratory relation, disturbances of similar origin occur where there is no association between the changes and the several acts of breathing. These disorders of the heart mechanism fall into three main categories. They are :— (1) Sudden and prolonged cessation of the whole heart beat, a condition so rare that it requires but a passing notice in this general survey. (2) Phasic variation of pulse rate, in which a retardation and subsequent gradual acceleration of the whole heart occurs ; the change is spread over ten, fifteen or more seconds and may be repeated regularly or may occur from time to time ; it is associated with the administration of heavy doses of drugs of the digitalis group, but may be seen apart from them (Fig. 7) ; it is a relatively uncommon type of irregularity. (3) An irregularity of the whole heart of mild degree, in which shorter and longer pauses are mingled indiscriminately. It is not infrequent, and is almost always combined with a general reduction of pulse rate. It may be found in quite young and apparently healthy children (Fig. 8) and is also encountered in young adults in whom no other cardiac sign is apparent. It is specially frequent in patients who have rheumatic heart disease and who are under the influence of digitalis; it is accentuated when the heart slows after it has quickened in response to exercise.

These sinus irregularities, like those which are related to respiration, are due to alterations of vagal tone.

The recognition of sinus irregularities.

Sinus irregularities are usually recognised with ease. It may be said that the great majority of pulse irregularities which occur before the end of the first decade are of this kind,

and most of them are respiratory. When there is the definite and stated relation to respiration, no further evidence is required; in most instances of sinus irregularity, this relation is present, but if it is absent, it becomes established if the breathing is deepened; a gradual waxing and waning of rate is always highly suggestive, if not conclusive. The radial beats and apex pulsations correspond; the heart sounds are simply modified according to the incidence of the ventricular contractions. The radial beats are full, and the apices of the several pulsations maintain an almost constant height in arterial curves (Fig. 4, 5 and 8).

The irregularity is abolished by any factor which notably increases the average pulse rate. Thus it disappears with exercise, with fever, or shortly after the administration of atropine.

The prognostic significance of sinus irregularities.

The commoner forms of sinus irregularity (excluding the prolonged and sudden cessation of the heart beat and the true phasic variation of pulse rate) are of little prognostic value. They are so frequently found in patients who present no other sign of cardiac disturbance, either at the original examination or subsequently, that they are to be regarded either as slight exaggerations of a normal phenomenon (respiratory irregularity) or as evidences of a mild and insignificant instability of tonic inhibitory nerve action.* Their importance lies chiefly in possible confusion with other forms of heart irregularity. They should not be allowed to influence the habits of those who exhibit them; neither do they suggest or require any special therapeutic measures.

* Occurring in children, this irregularity has obtained a grossly exaggerated and unenviable reputation, on account of its supposed relation to tuberculous meningitis.

Such are evidently the cases of irregular heart action that I have seen for years in cadets who have no sign of heart trouble: such cases have in time cleared entirely

CHAPTER III.

HEART-BLOCK.

Definition.

An abnormal heart mechanism, in which there is a delay in, or absence of, response of the ventricle to auricular impulses.

The nature of heart-block.

Under normal circumstances the ventricle depends, for its stimulus, upon impulses which are sent down to it from the regularly contracting auricle. Each auricular systole transmits a stimulus to the ventricle and this stimulus travels from auricle to ventricle along a narrow neuro-muscular tract, the auriculo-ventricular bundle. This band of tissue starts in the right auricle near the coronary sinus and proceeds forwards and downwards to the membranous septum of the ventricle (Fig. 1), where it divides into two main branches on either side of the septum. The main branches subdivide and are connected to the ventricular musculature through the complex network of cells named after Purkinje. The sequence in which the chambers of the heart contract is diagrammatically illustrated by Fig. 9. The black rectangles represent auricular (*A*) and ventricular (*V*) systoles.

When from any cause the function of the tissues uniting auricle and ventricle is impaired, a disturbance of this sequential contraction is engendered. The grades of disturbance, which human hearts manifest, are numerous.

There may be a mere prolongation of the interval which separates the commencements of auricular and ventricular systole (the *As- Vs* interval, as it is termed). Such a conduction defect is illustrated by Fig. 10 ; the thin lines become more oblique in the diagram, and a gap is left between the end of auricular and the beginning of ventricular systoles.

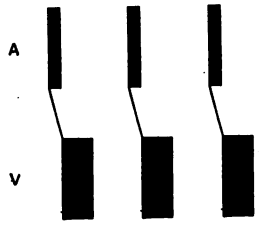

FIG. 9.

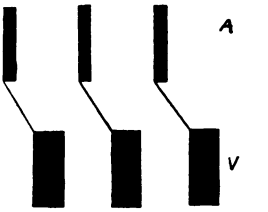

FIG. 10.

Fig. 9. A diagram representing the action of the normal heart. The auricle contracts first and transmits an impulse (the oblique line) to the ventricle. The ventricle responds and commences to contract immediately at the cessation of auricular systole.

Fig. 10. A diagram illustrating the earliest stage of heart-block. An interval appears between the end of auricular and the commencement of ventricular contraction. There is delay in the transmission of the impulse from auricle to ventricle (indicated by the obliquity of the line which joins the rectangles in the diagram).

Where the grade of heart-block is higher, the ventricle may fail to respond to occasional auricular impulses. Such is the condition when " dropped beats " are spoken of. This form of heart-block is rarely a simple phenomenon ; it is almost always complicated by variations in the lengths of *As- Vs* intervals over the period of disturbance. The relation of chamber contractions may be studied in Fig. 11. A " dropped beat " or ventricular silence produces a pause of exceptional length and this pause breaks the natural

rhythm of the ventricle. Where there is no associated variation in the *As- Vs* intervals, the length of the pause is necessarily equal to that of two regular pulse beats. Unhappily for interpretations, this is but rarely the case; the "dropped beat" is foreshadowed by a progressive increase of the preceding *As- Vs* intervals (cp. Fig. 11, *1*, *2* and *3*). Moreover, the *As- Vs* interval which follows the silence, is generally curtailed (Fig. 11, *4*). These two events shorten the long pause and consequently diminish the disturbance of the ventricular rhythm. The exact manner in which the changes happen are of importance and require closer study. Consider the first three *As- Vs* intervals of Fig. 11; as illustrated by the obliquity of the lines, the interval gradually widens, but it widens in a peculiar manner. *The*

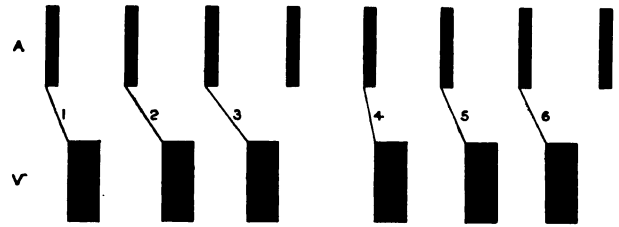

Fig 11. The next stage of heart-block, to which the term "dropped beats" is applied. Up to the point where the chief disturbance occurs, the gaps between the auricular and corresponding ventricular contractions widen out. The impulses travel to the ventricle with increasing difficulty. The fourth auricular contraction stands isolated, it yields no response; a ventricular contraction is "dropped." Following the ventricular pause, the *As- Vs* interval is short, for the tissues have rested, but it again widens as successive cycles follow.

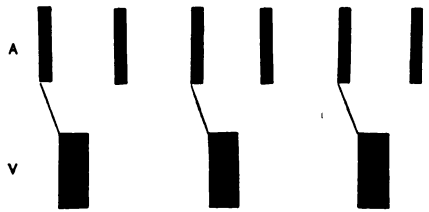

Fig. 12. A diagram of 2 : 1 heart-block, in which alternate ventricular beats are "dropped."

increase of the second interval over the first is greater than the increase of the third over the second. The result is a decrease of the interventricular period directly preceding the ventricular silence. *The ventricle quickens to the point of the disturbance.* The shortening of the *As- Vs* interval following the pause, and the subsequent prolongation of it, produces *a similar quickening of the ventricle after the disturbance.* The primary and secondary accelerations of ventricular rate, before and after the disturbance, offer a very helpful clue to the recognition of many cases of clinical heart-block.

As the grade of heart-block rises, and ventricular silences become more frequent, relatively simple ratios are established between the auricular and ventricular rates. When the ventricle beats at only half the rate of the auricle, because alternate impulses are ineffective, the condition is spoken of as 2 : 1 heart-block (Fig. 12). 3 : 1 and 4 : 1 ratios, in which each third or fourth auricular impulse alone yields a ventricular response, are sometimes encountered, but they are not common.

The mechanisms which have been described are all included under the term "partial heart-block."

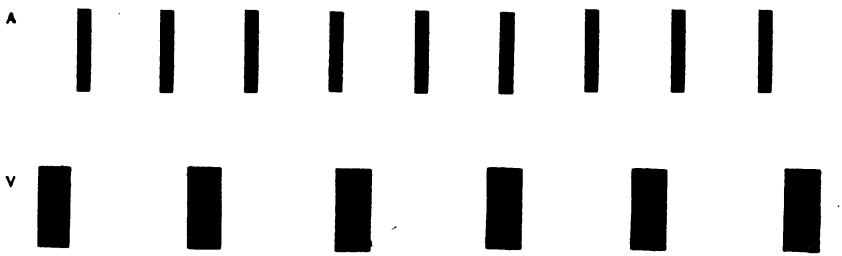

Fig. 13. A diagram of complete heart-block or dissociation. Both the auricle and the ventricle beat regularly, but at independent rates. The relative positions of auricular and ventricular contractions are very variable.

The final grade of heart-block is reached when no impulses are transmitted to the ventricle. When this happens, the ventricle, having completely lost the controlling influence of the auricle, beats in response to a slow and regular series of impulses which it builds up intrinsically. In "complete heart-block" or "dissociation" two entirely separate rhythms are maintained; one starts in and controls the auricle, the other originates in and controls the ventricle. The first has the usual rate, 72 per minute or thereabout, the last has an approximate rate of 30 to the minute. Though both are regular, the rhythms are quite independent (Fig. 13) and the systoles of auricle and ventricle fall with very varying time relations to each other.

Etiological and pathological associations.

Age. Heart-block may occur at any age. It has been observed in the newborn child and at almost all ages into the eighties and nineties. Its age distribution is settled by the age incidence of the diseases which produce it. Thus, it is especially prevalent amongst those whose hearts have been severely damaged by rheumatic fever or chorea, so that a special class of cases is grouped between the years 10 and 35. Senile affections account for another large group of patients who suffer from this cardiac disturbance; these patients are elderly. But the causation is so varied that no age is exempt. The distribution in my own series has been as follows:—

Age	10-20	20-30	30-40	40-50	50-60	60-70	70-80	80-90
Cases	7	6	3	5	2	3	4	1

Sex. As in other disturbances of the cardiac mechanism, heart-block is most prominent in the male sex. I may

illustrate this by a reference to my own series, in which 22 are males and 9 are females.

Heredity. A single instance of the supposed occurrence of several cases of heart-block in the same family has been reported, but has not been substantiated. It is highly improbable that heredity plays any direct part in the affection.

Relations to infective disease. Relatively, heart-block is not infrequent during the course of infective diseases, and of these I believe rheumatic fever holds the first place; the disturbance is usually temporary. The exact relation of rheumatic affections to acute and sub-acute inflammatory lesions of the heart is but imperfectly known, but there is a clear connection between the two. A number of instances of heart-block has been reported during the course of severe rheumatic fever and its complications, acute endocarditis and pericarditis. It is probable that the infection of the heart is rarely limited to its outer or inner layer; there is a growing belief that the middle layer or myocardium is also frequently involved. My own experience leads me to think that heart-block is almost, if not quite, a common accompaniment of acute or subacute rheumatism of the heart, for I have seen several cases recently in which, during the course of rheumatic fever involving the valves or pericardium or both, the patients exhibited dropped beats or partial heart-block in its several grades. In other instances, temporary heart-block has appeared during short febrile attacks in patients who have been previously affected by rheumatic fever. It is certain that being transient it is often overlooked.

Of other acute affections which should be mentioned are those of the more active pus organisms, and also those of severe diphtheria, influenza, typhoid and pneumonia, though the frequency of heart-block in these conditions is still unknown.

A very large proportion of the reported cases of *chronic* heart-block and of those which have come under my own observation, have belonged to two groups; the disorder has been found subsequent to single or repeated attacks of rheumatic fever, or has been the direct result of syphilis. Whether of rheumatic or syphilitic origin, heart-block is generally but an expression of a widespread affection of the heart muscle in these patients, though the lesion may be confined to, or may fall most heavily upon, the tissues which establish functional connection between the auricle and ventricle. In a fourth of the cases in which direct examination of the hearts has been obtained the lesion has been gummatous. From my own series of 31 cases, 4 gave a history of venereal infection and 10 a history of rheumatism.

The relation of heart-block to rheumatism in chronic heart affections is a peculiar one. The heart-block is often dormant or is detected only by exact instrumental methods; it is often unmasked by the administration of drugs of the digitalis group, for the higher grades of heart-block are produced from the lesser by these poisons.

Relation to chronic degenerative processes of more obscure origin. A very large number of the reported cases of heart-block have been in elderly people, and observation has shown that the damage responsible for the disturbance has been part and parcel of a more or less widespread change, either in the heart itself, or in the heart and its vessels. A number of the lesions can undoubtedly be traced to syphilis or rheumatism, but the cause of a still larger number is obscure. Chronic inflammation, fibrosis, atrophy, calcification or fatty degeneration of the tissues, associated or unassociated with disease of the coronary arteries, are amongst the most frequent causes.

Heart-block as a result of digitalis administration. I have already referred to the unmasking of dormant heart-block in rheumatic heart disease. When digitalis or its allies, strophanthus and squills, are given in toxic doses to young patients who have rheumatic hearts, it is not uncommon to observe the severer grades of partial heart-block as a result. And it is known that in most of the cases which react in this manner a slight defect in the conduction of impulses from auricle to ventricle was present before the drug was taken. The added effect is probably due in some measure to the action of digitalis upon the vagus nerve, for it can be partly removed by atropine.

Heart-block can be induced in experiment by stimulation of the vagus, and efforts have been made to establish a clinical group in which the heart-block is attributable to disturbance of innervation. Up to the present time, there is no very clear record of the initiation of even a temporary disturbance of this character by vagal impulses; though, as I have stated, a pre-existing tendency may be exaggerated in this manner; while if the higher grades of persistent heart-block are ever due to derangement of vagus action, such a causation is so rare that it scarcely comes within the practical field.

Morbid anatomy. It is in the main bundle, or in its auricular attachment, that most of the lesions responsible for heart-block have been described. The kind of lesion has been spoken of already. Gummata, chronic inflammatory processes and their accompaniments, fibrosis, atrophy, and calcification are most frequently found. Examples of tumours (fibroma and endothelioma) affecting these special tissues have been recorded.

Ulceration invading the bundle, acute inflammation evidenced by deposition of leucocytes or parenchymatous

degeneration of the bundle are the common lesions in hearts which have been damaged by acute infections.

The recognition of heart-block.

The disorders of the heart's mechanism caused by heart-block, in its several grades, are readily recognised by the exact graphic methods provided by the polygraph and galvanometer. The efficacy of these instruments and the certainty of the analysis must be evident, for heart-block produces derangement of sequence in the contractions of auricle and ventricle, and the polygraph and galvanometer supply separate records of the systoles of upper and lower chamber. Therefore, a comparison of the onsets of the several systoles is relatively simple when these recording devices are employed.

But I speak to those to whom the special method is not available, and I hope to show that heart-block can be recognised in many of its grades by simpler means. It will be necessary to treat each form of mechanism separately and, in this instance, to refer especially to exact measurement of the arterial pulse pauses. In many forms of irregularity such measurement is not necessary, though it may be expedient; in the disturbance produced by heart-block it is usually essential.

Often the earliest manifestations of heart-block consist in a **widening of the As-Vs interval** (see page 17); this defect can rarely be identified by ordinary clinical means; yet it may be responsible for two physical signs. It may not be known to everyone that auricular systole produces a distinct though muffled sound, and that while this sound is inaudible when the heart's mechanism is normal, it is frequently heard when the auricular and ventricular systoles are sufficiently

separated. A slight widening of the *As- Vs* interval may lead to a reduplication of the first heart sound; a more pronounced widening may result in a double second sound, for the auricular systole may fall in early diastole.

The second sign is confined to cases of mitral stenosis and is of similar origin; in these patients the systole of the auricle is the cause of the murmur which characterises the valve lesion. Contraction of the auricle at an abnormal instant in diastole is accompanied by a murmur and thrill which replace the customary presystolic events. When the pulse is regular, apical thrills or rough murmurs, confined to mid- or early diastole, are physical signs which should suggest not alone stenosis but also the beginning of heart-block.

Single dropped beats are not difficult to identify. Take the case where a pulse, which seems otherwise regular, is interrupted by an occasional pause of unusual length, while examination of the apex beat reveals neither movement nor sound in the pause. If the pause is not associated regularly with the phase of expiration (see page 12) it can be attributed to a failure of the customary response of ventricle to auricle. The length of the pause in radial tracings may be exactly equivalent to that of two rhythmic beats. More frequently (as in the radial pulse tracing of Fig. 14) it is distinctly short

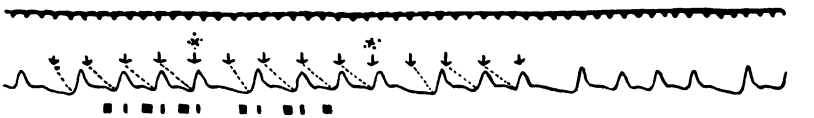

Fig. 14. A pulse curve showing "dropped beats." The arrows, which represent the positions of the regular auricular contractions, have been accurately determined in this and subsequent figures by means of polygraphic curves. The heart sounds are shown diagrammatically. The arrangement of the pulse beats depends upon the lengths of the *As- Vs* intervals and upon the failure of response at the points marked by asterisks. Note the widening of the *As- Vs* intervals and accompanying increase of pulse rate before and after each dropped beat.

of this, and is preceded and succeeded by slight pulse quickening. The nature of these phenomena has been considered already (page 18), and the mechanism is indicated in the present figure by means of the arrows which show the points at which the regular auricular systoles fall. Responses to the auricular contractions marked by asterisks have failed.

When dropped beats are more frequent, the irregularity takes the form of that seen in Fig. 15. Here each third or fourth impulse miscarries, and the heart and pulse beats are grouped in twos and threes. Had we not the termination of this curve, the analysis of the first half could not be completed, for the picture is identical with that produced by premature contractions. The clue to the true interpretation is given by the lengths of pauses *1* and *3*; they are equal. These are the opening beats of two groups, the first of two, the second of three pulsations. The long pause which follows each group is of constant length; it has been produced by a constant mechanism. If the pause which follows the first group were attributable to prematurity of pulse beat *2*, a similar pause would be expected after pulse beat *4*. It does not occur, and we recognise in *3* and *4* the acceleration of pulse rate which precedes or follows an unusual pause resulting from heart-block.

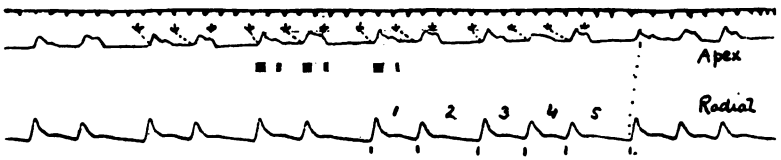

Fig. 15. Curves of heart's apex beat and pulse from a patient in whom ventricular responses failed frequently.

2 : 1 heart-block is to be suspected in any patient in whom the ventricle beats regularly and where the rate lies between 40 and 50 contractions a minute. A sudden and exact halving of *ventricular* rate is always most suggestive. 2 : 1 heart-block is usually an unstable condition, the ventricle quickening from time to time, and these changes in the

frequency of its response to auricle disclose the nature of the whole disturbance.

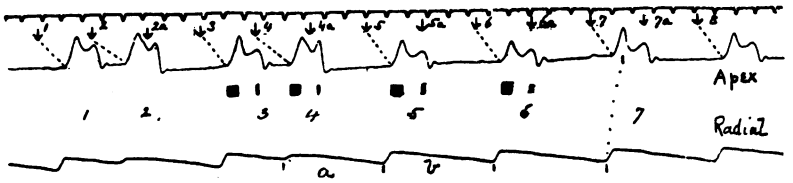

Fig. 16. Curves of heart's apex beat and pulse, taken during the passage of the mechanism from a condition of frequent "dropped beats" to 2 : 1 heart-block. The rate is reduced to exactly three-fourths the original at the change.

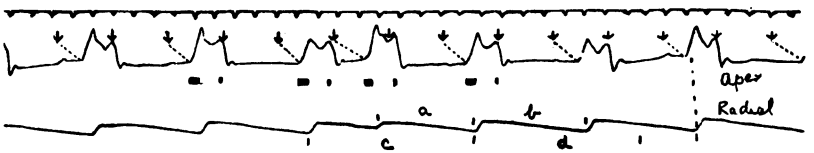

Fig. 17. Curves showing the interruption of a period of 2 : 1 heart-block by a single response of the ventricle to one of the series of alternate impulses which usually yield no ventricular contraction.

The transition between 2 : 1 heart-block and an arrangement previously studied, namely, the loss of each third response, is shown in Fig. 16. A bigeminal or coupled action of the ventricle passes over into a slow regular action. The features which proclaim heart-block in this curve are the increase in the length of pause from *a* to *b*, and the exact reduction of rate to three-fourths. The lengths of the several pauses are understood by examining the positions of the auricular systoles which have been indicated by arrows drawn on the curve. Systoles *2a*, *4a*, *5a*, *6a* and *7a* do not affect the ventricle; and where the ventricle is silent an unusually lengthy pause is found. The arterial pause *a* is brief as compared with *b* because the auricular impulse *4* takes longer to reach the ventricle than does impulse *5*. Disturbance of a 2 : 1 period is shown in Fig. 17. An early contraction of the ventricle is followed by a pulse pause *a* which is shorter than *b* and the succeeding pauses. The reason of this shortening has been explained in the description of the last figure. In Fig. 17 heart-block is also evidenced by the fact that the total duration (*c*) of the two short beats is equal to one and a half times the duration of the longer beats (period *d*). In other words *c* and *d* correspond to three auricular cycles.

In mitral stenosis partial heart-block is often characterised by peculiarities of the murmurs. They are often extremely complex. Where 2 : 1 heart-block is present, two thrills and two diastolic murmurs may accompany each

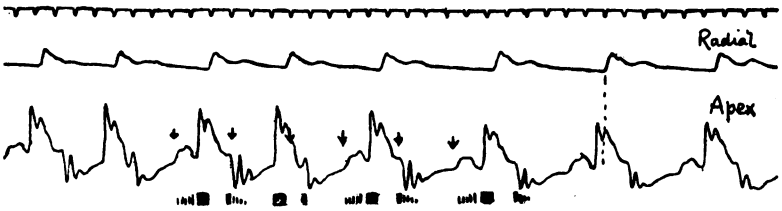

Fig. 18. Arterial and apex curves from a case of mitral stenosis, while the mechanism passes from a condition of dropped beats to 2 : 1 heart-block. Note the arrangement of the diastolic murmurs and their dependence upon auricular contractions.

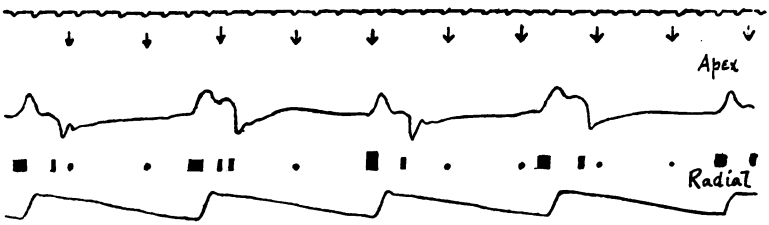

Fig. 19. Apex and radial curves in a case of complete heart-block. The heart sounds are modified by the auricular contractions, which are faintly audible. Where auricular and ventricular contractions begin together the first sound is exaggerated. The pure auricular sounds are shown as dots.

ventricular cycle. The meaning of the phenomenon will be obvious if it is remembered that the thrill and harsh murmur of mitral stenosis are produced by auricular systole and that in 2 : 1 block the auricle contracts twice as frequently as the ventricle. A more complex arrangement of murmurs is

shown in Fig. 18. The ventricle beats at first in couples, and at such times the murmur occurs before the first and after the second sound of the first beat of a couple; the second beat of the couple is accompanied by no murmur, for the single auricular contraction in its neighbourhood falls with that of the ventricle and no blood is forced through the stenosed orifice. Over the last portion of the curve 2 : 1 heart-block is present, and each cycle is accompanied by presystolic and early diastolic murmurs.

In complete heart-block the action of the ventricle is phenomenally slow; almost all hearts which beat at rates of 35 and under are affected in this manner. The rhythm is generally quite regular. Each ventricular beat is accompanied by a first and second sound, and in addition very faint muffled sounds are heard in the long diastoles. These are due to auricular systoles. A sign which is characteristic and often present, is a modification of the first and second heart sounds from beat to beat. When the auricular and ventricular contractions begin together, the first sound is intensified, and when they fall almost together the first or second sound may be reduplicated (Fig. 19). Evidences of the relatively rapid auricular contraction are generally seen in the neck; small and regular pulsations (Fig. 20, *a* waves) are shown by the jugular veins between the beats of the carotid (*c* waves). From time to time a prominent venous pulsation (Fig. 20, *a*/*c*) accompanies the intensified first heart sound, when auricular systole coincides with that of the ventricle and when as a consequence the blood cannot be driven forward out of the auricle. A periodic waxing and waning of the venous pulsations, independent of respiration, is always highly suggestive of the condition. Traces of auricular pulsations upon the arterial curves are also evident in most of the patients from whom full pulse excursions can be obtained

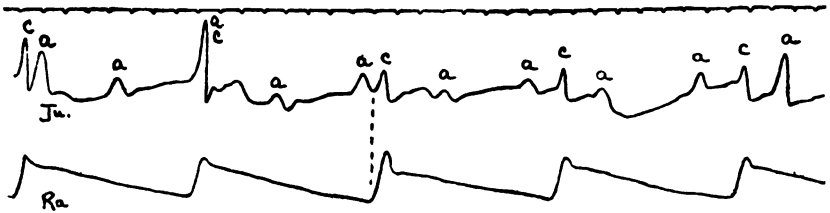

Fig. 20. Curves from the neck (*Ju.*) and radial artery in a case of complete heart-block. There are three pulsations in the neck during each radial cycle. Two of each group of three neck waves result from auricular contractions, *a*, while the third is the result of ventricular systole, *c*. When *a* and *c* fall together an exaggerated wave is produced and is visible as such in the neck. It is due to discharge of the auricular contents into the veins.

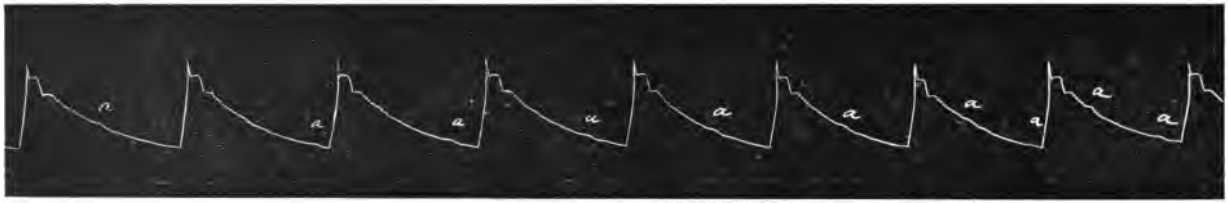

Fig. 21. A sphygmographic curve from the radial artery in a case of complete heart-block. As often happens, signs of auricular systoles (*a*) are visible in the curve and are easily recognised by their regular change of position as they are traced from one radial cycle to the next.

(Fig. 21). Where, as in the accompanying figure, the little waves on the downstrokes of the regular pulse beats show a gradual and orderly change of position, moving steadily away from the succeeding radial upstroke, the presence of complete heart-block is certain.

Effects on the circulation and the general symptomatology.

The symptoms presented by patients with heart-block are divisible into two groups. On the one side are the symptoms which are the special appurtenance of the condition itself, and on the other are those which result from co-existing disease in other portions of the heart. For disease is rarely limited to the bundle, and generally heart-block is but a local manifestation of a more widespread process; the local lesion is often accidental. The effects of a lesion which transects the bundle differ from those of a similar lesion in another portion of the musculature in one chief respect; the damage gives rise to obvious disturbance; there is no second strand which may fulfil the functions of that which is destroyed, whereas a defect in the general mass of muscle is hidden by the response of the remaining tissue. As in disease of the nervous system, where large areas of tissue may be lost without gross signs of damage, but where a minute morbid focus in a given situation gives rise to obvious and profound disturbance; so it is with the heart. It is necessary to emphasise the fact that heart-block is usually an indication of a far graver condition than simple transection of the bundle; it is a sign of wide or universal invasion of the myocardium.

The symptoms which result from affections of the whole heart musculature do not lie within the scope of this book; but it is important to recognise that the presence of heart-block demands an exhaustive study of the subject in whom it appears; in all instances special attention should

be directed to the fitness or otherwise of the heart as a whole. And this caution is not limited to heart-block, it applies to all departures from the normal mechanism.

The special symptomatology of heart-block may be conveniently approached from two standpoints.

Heart-block of high grade is accompanied by a reduction of the rate of the heart beat, often to a half its former rate. What is the effect of this retardation of heart rate upon the circulation, and what are the results of the lessened nervous control of rate which often accompanies it? It is certain that thereby a serious burden is imposed upon the efficiency of the heart as a pump; but nothing is more remarkable than the accommodation of the cardiovascular system to conditions which diverge widely from the normal ones. Dissociation of auricles and ventricles, and the consequent establishment of a slow ventricular rhythm, is followed by some degree of ventricular hypertrophy. Undoubtedly, this increase in the mass of the ventricular muscle compensates in a measure for the loss of co-ordination and of the natural rates. During the long diastoles the blood is driven from arteries to veins and a low diastolic blood pressure results; but the blood pours equally fast from veins to heart, whose efficient chambers, receiving the extra load, expel it into the arteries. Fullness of pulse and high systolic pressure (170-200 mm. Hg.) consequently characterise the arterial system when in persistent heart-block there is no lack of healthy cardiac tissue. As evidences of the adaptability of the circulation as a whole to the new conditions, I may cite the case of a patient in whom, judging from the signs and symptoms, the damage to the muscle mass is but little. The patient, a man of 33 years, is known to have had a heart rate of 30 to 35, with occasional accelerations to 48, for 11 years. He is the subject of complete heart-block. There is a little hypertrophy of the heart, but no subjective symptoms. He leads a very

active business life, and passed in the street would be judged a perfectly normal and healthy individual. There is no circulatory embarrassment, even after strenuous exertion; he prides himself upon his "sprinting" power and has raced during the past few years. An instance of this kind offers a partial answer to the original questions; the slow pulse of heart-block and the absence of regulation of rate do not disable an otherwise healthy heart from performing its full work. In hearts more profoundly affected, the extra burden is less readily borne, but in these it is not easy to dissociate the effects of the new mechanism from those of disease of the remaining muscle.

In the second place, heart-block is responsible for a group of symptoms which arise as a direct result of excessive slowing. Reduction of pulse rate beyond certain limits, or the cessation of the arterial flow for a certain time, is accompanied by grave disorders of nutrition, and the brain is an early and anxious plaintiff. The patient, who exhibits marked pulse slowing in conjunction with fits, falls into the category of Adams-Stokes' Syndrome. The higher grades of heart-block, whether of persistent heart-block in which ventricular responses are frequently missed (2 : 1, 3 : 1 ratios, etc.), or of complete dissociation, are frequently accompanied by temporary periods of excessive pulse slowing or by cessation of the ventricular systole for prolonged intervals. The cause of the change in ventricular rate, for the auricles continue to beat at the usual or at an enhanced rate, is not fully understood, and I do not propose to consider it further. The symptoms presented by the patient are intimately dependent upon the degree of heart slowing or upon the duration of isolated periods of asystole. When the pulse falls to 8 or 20 beats per minute, unconsciousness supervenes; suspension of the mental functions is also produced by a single period of asystole of from 3 to 7 seconds

duration. Patients who suffer from the higher grades of heart-block commonly give a history of brief attacks of giddiness, fainting, temporary loss of consciousness and its dependent accidents. Seen in mild attacks, the subject of them is pulseless and momentarily pale. In severer seizures, where the pulse ceases for 15 seconds or more, there are additional phenomena. The blood is dammed back in the venous system, increasing pallor has cyanosis added to it, the breathing deepens and becomes stertorous; twitching of the face and upper limbs eventually occurs. The convulsive fit rarely spreads beyond the named area, but it may become more generalised. Urine is not passed, neither is the tongue bitten during the attacks. In most cases the condition is readily recognised by the absence of signs of ventricular activity and by the presence of rapid undulations in the veins of the neck, signifying activity of the right auricle. Unexpected death is a by no means uncommon accident amongst the affected, but considering individual attacks it is rare. Death occurs after a period of status epilepticus in a number of the patients, and the status consists of repeated seizures of the forms described.

As a rule the patient has no warning of an impending syncopal or epileptic attack; though on occasion he or his medical attendant may be informed of the approaching danger by a change in the heart's action, for example, by the occurrence of further ventricular slowing. The sensations of the patient at the commencement of long seizures are usually similar to those accompanying a brief cessation of the heart beat, and consequently do not properly constitute an aura.

The prognosis.

Heart-block in itself does not kill; those who suffer or have suffered from it mostly die with the usual symptoms of general heart failure. Let me be clearly understood in this

statement. Heart-block and the Adams-Stokes' syndrome are not synonymous terms; the majority of patients who exhibit heart-block never have fits. Lesser grades of heart-block are common in conjunction with rheumatic heart disease and as a rule they produce no symptoms. Moreover, the disturbed mechanism is not of necessity directly fatal even in chronic heart-block of high grade.

The prognosis in heart-block has to be dealt with from several points of view. In the first instance, let us consider the milder grades of heart-block, such as are associated with rheumatic heart disease (prolonged *As-Vs* intervals or "dropped beats"). Where such heart-block is *persistent*, there are usually a number of physical signs in addition to those dependent upon the heart mechanism; they are the signs of heart disease, muscular or valvular, and in its several and universally recognised forms. Heart-block is often the least prominent symptom in these cases; they are often to be classed as mitral stenosis. The only question that I raise is as to the manner in which heart-block affects the prognosis in these cases. It should be regarded as an evidence of myocardial damage, not necessarily limited to the bundle, but probably diffused throughout the heart. My experience of such cases is that they are serious; in fact, most of those I have seen are dead, though they did not die of heart-block. But *temporary* heart-block of mild grade is not uncommon during the febrile attacks to which rheumatic patients are liable; it occurs also in pneumonia and typhoid. The appearance of this abnormal mechanism is of great importance, for it is often the sole sign which indicates that the myocardium has been damaged. Whenever it complicates an acute infection it consequently increases the gravity of the prognosis; at the same time it should be understood that the normal mechanism is usually recovered. Occurring as an accompaniment of fever in a patient who has rheumatic heart

disease, it should be regarded as an outward sign of an isolated injury which, if often repeated, eventually so weakens the muscle that life is no longer supported.

Where the higher grades of heart-block are present, the prognosis is based upon two chief considerations. The general evidence of the integrity and fitness of the muscle as a whole should be weighed first. The fits, especially their frequency and severity, are next taken into account : a number of the patients are free from them ; others are in constant peril ; and it is not easy, nay it is often impossible, to predict the ultimate effects of syncopal attacks or severer crises in a given case. Those patients especially, who have progressive lesions, and those in whom partial is eventually converted to complete and permanent dissociation, must pass through a time of particular danger ; for, during the passage from one mechanism to the other, fits are very common and the period of passage may not be a short one. It is useful to remember also that those who have partial heart-block are more prone to fits than those in whom the obstruction is complete. Uncertain in both incidence and effects, the fits always dictate a cautious prognosis.

Regarded in its entirety, persistent heart-block of high grade is a grave condition. It is usually complicated, and then a few years generally close the scene. Nevertheless, some, and especially the younger patients, survive for many years, in comparative and absolute comfort. They are those in whom the mass of heart muscle is comparatively healthy and in whom fits are rare or absent.

The treatment.

Persistent heart-block of the milder forms requires no immediate treatment, but is an indication for repeated examination of the patients who show it. As such patients usually require treatment for the general condition of the

heart, constant supervision is not difficult. They often require digitalis medication, and this will frequently increase the grade of block. But the increase of block should not deter digitalis administration for the relief of dilatation, dropsy or other symptoms; nor is it in itself detrimental; the drug or its allies may be given without restraint and often beneficially.

When the *abrupt onset of partial heart-block* is observed, it is, as I have said, an index of active mischief. The patient should lie up or remain in bed and should be thoroughly searched for the provocative cause, which, when found, is attended to. The acute infections are suitably treated. Rheumatic patients are treated wtih salicylates, and scrupulous attention is paid to the hygiene of the mouth and throat. If, after the subsidence of remaining symptoms, the block remains and persists for several weeks, the patient is treated along the lines indicated in the previous paragraph. Heart-block in itself does not call for rest in bed or other interference, though a suspicion of an active or progressive lesion does.

The *higher grades of heart-block* are usually chronic and stationary and the habits of the patient should be governed by his general fitness. Most patients of this class are up and about, and are able to undertake many of their ordinary duties; yet it is only in exceptional instances that real bodily activity is either possible or permissible. Here again a suspicion that the lesion is active or progressive calls for rest and careful watching. A history or sign of syphilis constitutes an imperative demand for thorough and appropriate treatment, and in some cases success has attended the administration of mercurials and iodides.

All those who have *fits* should be warned of the danger which they run from falls during fits, if they do not appreciate it fully. Not a few have lost their lives by falling heavily and suffering mortal injury. In many cases the fits occur in

groups, and additional precautions will be required until such attacks cease. Most patients have brief warnings of the onset of unconsciousness, and, if advantage is taken of them, less risk is incurred.

A careful enquiry for causes predisposing to the fits may elicit a history of gastro-intestinal disturbance or over-exertion, upon which it is well to act.

For the fit when it is present, I know of no remedy which is of avail to increase the pulse rate and restore the unconscious patient. A number of drugs have been administered with this end in view, and the list includes oxygen, strychnia, strophanthine, digitalin and amyl nitrite. They appear to have no appreciable effects. Atropine is said to have abolished fits in isolated instances. As a rule it is contra-indicated.

CHAPTER IV.

PREMATURE CONTRACTIONS.

Definition.

Responses of the heart to new and isolated impulses formed in the musculature; contractions which occur before the anticipated time and which consequently disturb the normal order of the heart's mechanism.

*The nature of premature contractions.**

A clear and full appreciation of an abnormal heart mechanism can be attained only by those who are perfectly familiar with its normal action. The orderly sequence of muscle movements, which constitute the normal heart beat, is propagated, as I have already stated, from a single impulse having its birth in the sino-auricular node. The contraction, starting from the mouth of the superior vena cava, travels rapidly through the auricle, reaches the auriculo-ventricular node and traverses this node and the bundle which is its continuation; it is distributed in an orderly manner amongst the mass of ventricular fibres in which it ends. The normal rhythm of the heart consists of a regular sequence of such beats, so that auricular and ventricular contractions fall with the proper time relations to each other. Each stimulus elaborated at the sino-auricular node requires a certain time

* I use the term premature contraction in preference to "extrasystole," a name which has been and still is employed to designate the same abnormal beat.

of preparation, and this time of preparation is very constant under definite and given circumstances. It is relatively long, reaching nearly two-thirds of a second when the heart is beating at a normal rate. Indeed it is the time of impulse preparation which controls the rate of a normally beating heart. A second characteristic of physiological impulse formation is regular repetition. Each impulse belongs to a regular or rhythmic series.

The premature or pathological contraction differs from the physiological in two fundamental respects. The impulse which gives rise to it is formed at a phenomenally rapid rate. It is to this quality that the pathological contraction owes its prematurity. The absence of a definite tendency for the pathological impulse to repeat itself constitutes its second distinctive feature, and upon this character the usual isolation of the pathological contraction depends. Premature contractions originate abruptly, and may spring from the auricle, from the ventricle, or from the tissues which unite these two contractile structures. For ordinary clinical purposes it suffices if we remember the two chief classes of premature beat, the auricular and the ventricular.

If, while the heart chambers are beating in a normal and sequential manner, a pathological impulse arises in the ventricle, the ventricular beat which it awakens will fall before the anticipated point in the rhythmic series; whence comes the term "premature contraction." It disturbs the sequence of ventricular contractions in a definite manner. Excepting the premature impulse, the ventricle is absolutely dependent for its stimuli upon the impulses which descend to it from the auricle. Consequently, after the disturbance produced by a single premature beat, the ventricle rests until a rhythmic auricular impulse reaches it. If the accompanying diagram (Fig. 22) is studied, it will be seen that for the first three cycles the ventricle follows the auricle in contraction; a

premature beat (*p*) is then interposed and as a consequence the next auricular impulse, represented by the broken line, arrives while the ventricle is already contracted. Being

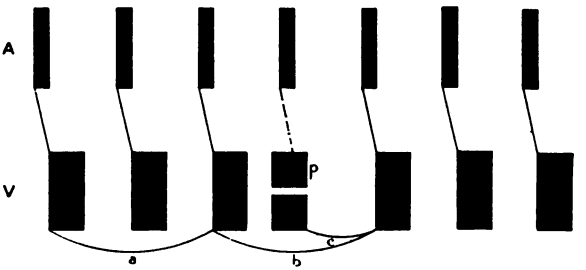

Fig. 22. A diagrammatic representation of the disturbance produced by a premature ventricular contraction (*p*). The auricle beats regularly throughout. The ventricle responds to six auricular impulses. The impulse of the central auricular systole is lost, for it falls while the ventricle is in premature systole. The abnormal origin of the ventricular beat is indicated by the break in its centre. Note the equality in the lengths of periods *a* and *b*. *c* is the compensatory pause.

in contraction the ventricle shows no response, its muscle is in the "refractory" state. The dominance of auricular impulses is reasserted during the succeeding cycle. Thus, the disorder is controlled by the fundamental heart rhythm which proceeds, unheedful of the disturbance. The ventricular contractions, subsequent to the disturbance, fall at points which may be accurately anticipated; the period of the disturbance (*b*) is exactly equivalent to the length of two complete cycles of the normal rhythm (*a*). The pause which follows the premature ventricular beat is long; the ventricle is waiting. The length of the pause (*c*) is such as to compensate for the brevity of the pause which precedes, consequently it is spoken of as the "compensatory pause."

When a premature impulse originates in the auricle the order of events is different. The premature contraction of the auricle, which it calls forth, is followed by a similar and parallel disturbance in the ventricle (Fig. 23), for the

ventricle responds to each auricular contraction wherever such contractions are placed in a series. In all but exceptional instances too, there is a disturbance of the fundamental heart

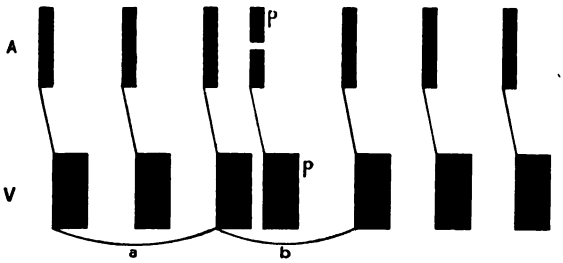

Fig. 23. A diagrammatic representation of a premature auricular contraction. The auricular rhythm is disturbed by the abnormal auricular beat (*p*); the disturbance in the ventricular rhythm is parallel, for each auricular systole yields a ventricular response. The rhythm of the whole heart is dislocated, the period *a* is longer than the period *b*.

rhythm; the premature contraction (*p*) is followed by a long pause, but the whole period of the disturbance (*b*) is not equivalent, as in the case of the premature ventricular beat, to two full cycles of the normal rhythm (*a*).

Etiological and pathological relations.

Age. In my own series the age limits between which premature contractions have been observed are 4 to 91. During the first decade they are extremely rare.* Their incidence in an age table is actually heaviest between 50 and 70 years; if the age distribution of the populace is considered in conjunction with this fact, it becomes evident that essentially they are a phenomenon of advancing years.

Age distribution of premature beats in 90 cases.

Age	0-10	10-20	20-30	30-40	40-50	50-60	60-70	70-80	80-90	90-100
Auricular	0	2	3	3	4	2	6	1	0	1
Ventricular	1	13	7	5	10	15	13	4	0	0
Combined	1	15	10	8	14	17	19	5	0	1

* The solitary instance, a child of 4 years, included in the table, was specially brought to my notice by Dr. Clive Riviere, who discovered it while examining school children.

Sex. Premature contractions are much commoner in men than in women. In 104 subjects the sex distribution was as follows :—

Sex	*Male.*	*Female.*
Auricular	15	10
Ventricular	54	25
Combined	69	35

Associated conditions and provocative factors. It should be remembered that any statistics compiled to show the relations of premature contractions to associated conditions and infections, suffer from a defect. Those cases which exhibit frequent and persistent premature beats preponderate in the tables; for under these circumstances they are conspicuous, while if they are scarcer they may often fail to attract attention. It is probable that the majority of people who live to middle life or advanced years are affected in this manner at some time or another. Amongst patients who attend out-patient departments or are admitted to the wards of general hospitals, frequent and persistent premature contractions are most common in those who exhibit definite symptoms and signs of cardiac disease. They are often found in association with aortic imcompetence and mitral stenosis ; an even larger number of curves are collected from patients who present signs of degenerate heart muscle, as evidenced by dilatation and symptoms of muscle insufficiency in the absence of gross valve lesions. In yet another large group of patients, no evidence of functional impairment of the heart, leaving the irregularity out of consideration, can be discovered.

Premature auricular contractions.

Cardiac group.		*Remainder.*	
Myocardial degeneration	9	Bronchitis and emphysema	2
Mitral stenosis	4	Pulmonary tuberculosis	2
Aortic stenosis	1	Dyspepsia	1
		Lumbago	1
		Exophthalmic goitre	1
		Apparently healthy	1
	14		8

Premature ventricular contractions.

Cardiac group.		*Remainder.*	
Myocardial degeneration ..	18	Tuberculosis (lungs and pleura)	5
Aortic disease	12	Bronchitis and emphysema ..	2
Mitral stenosis	12	Gallstones	1
Angina pectoris	5	Gangrene of toes (senile) ..	1
Bright's disease and granular kidney	5	Epilepsy	1
		Lipoma of neck	1
Arteriosclerosis	2	Fractured skull	1
Acute endocarditis	2	Abdominal tumour	1
Aneurism	1	Exophthalmic goitre	1
		Gastric ulcer	1
		Dyspepsia	1
		Apparently healthy otherwise	2
	57		18

Of the factors which appear to be predominantly associated with them, gross lesions of the heart stand first. Otherwise an inquiry into the habits, history and state of the patients throws but an obscure light upon the causation. A history of rheumatic infection is certainly common, it was present in one-third of the cases in my series. In young adults, excessive tobacco smoking is recognised as an exciting cause of their temporary appearance. Digitalis and its allies are not uncommonly responsible, when the patient is under the full influence of these drugs. There are also clinical associations between premature contractions, raised arterial pressure and digestive disturbances, but these are not fully understood at the present time.

Many things affect the frequency of premature contractions. Fatigue, subsequent to exertion, is provocative in those who are predisposed. The influence of heart rate is especially noteworthy. Hearts beating at 100 per minute and over are not often disturbed, and premature contractions are very rare when the heart rate exceeds 120. Fever usually rids the pulse of this form of irregularity, and so also does any other cause which notably accelerates the pulse rate. Thus they are abolished during exercise and for a short period afterwards, but during the period of slow heart action which often follows exercise, they are frequent. As we

shall subsequently see, this knowledge may often be used advantageously to induce premature beats in patients predisposed to them. Suspension of respiration for a period compatible with comfort often suffices. The pathological beats are in evidence either in the apœnic stage or shortly after the resumption of respiration. No factor is more potent than posture. Patients, who exhibit numerous premature contractions while standing, may soon lose them in recumbency, and this despite a slight decrease of heart rate in the last position. In other patients, pressure upon the abdomen may abolish them.

The recognition of premature contractions.

The work accomplished by premature beats is small, because the periods of rest that precede them are short. They may or may not *raise the aortic valves.* Accompanying the premature beat, a feeble pulsation or a prolonged pause is noted in the arterial pulse; auscultation reveals early first and second sounds when the aortic valves are forced, but only an isolated and premature first sound if the ventricular pressure fails to top the arterial. The consequent grouping of sounds in threes and fours is comprehended when the nature and degree of the corresponding arrhythmia are discerned. The common arrangements of pulsations and sounds are described in the following paragraphs, and are illustrated by the accompanying diagram and tracings.

In the succeeding paragraphs I have sub-grouped the symptoms according as the premature beat (α) raises or (β) fails to raise the aortic valves.

1. When a systole of a regularly beating ventricle is replaced by a premature beat, this abnormal contraction is accompanied by an early apex thrust and by (α) a weak arterial wave and two extra sounds, which together with those of the preceding rhythmic beat form a group of four

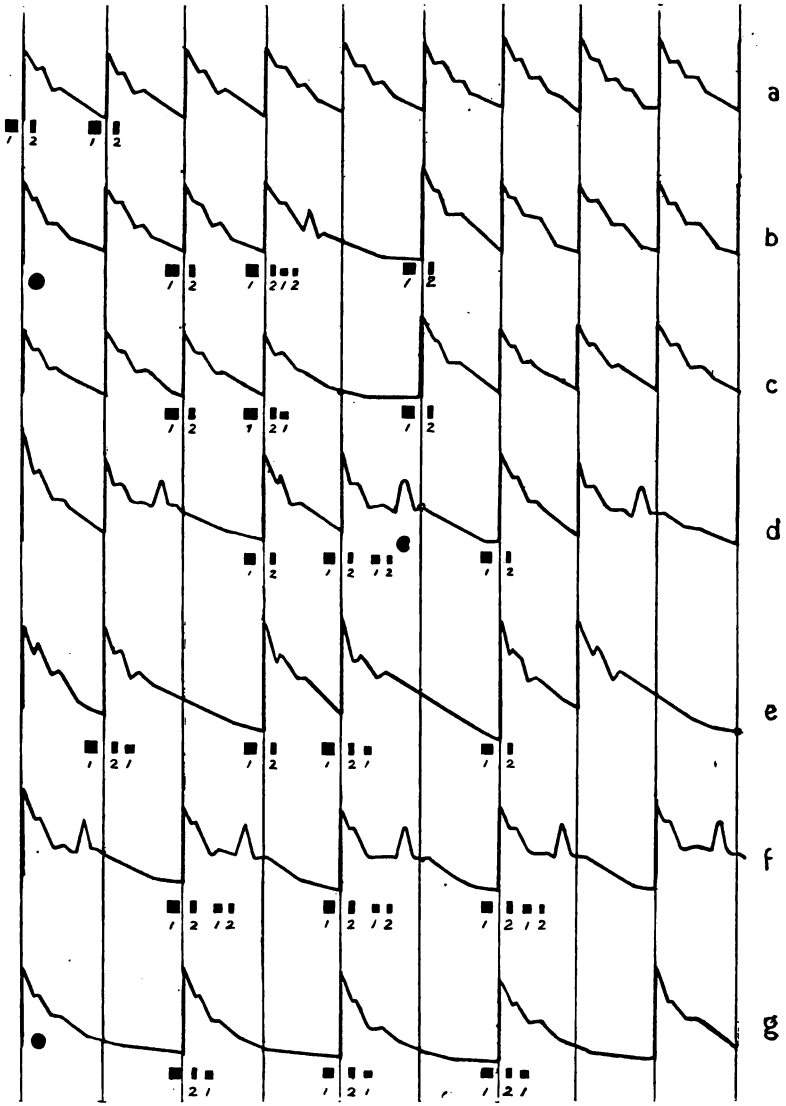

Fig. 24. A diagram showing common disturbances of the arterial pulse and heart sounds when premature ventricular contractions are present.
(*a*). Normal rhythm ; (*b*). Occasional premature beat, which affects arterial pressure ; (*c*). Occasional premature beat, which fails to affect arterial pressure ; (*d*). Premature beat replacing each third normal beat and affecting arterial pressure ; (*e*). Premature beat replacing each third normal beat and failing to affect the arterial pressure ; (*f*). Premature beat replacing each second normal beat and affecting the arterial pressure ; (*g*). Premature beat replacing each second normal beat and failing to affect arterial pressure. The heart sounds occur in groups, and the groups are of four or three, according as the aortic valves are raised or remain at rest when the premature beat occurs.

(Fig. 24 *b* and 25); or by (β) an intermission in the arterial pulse and one extra sound, forming with the sounds of the preceding rhythmic beat a group of three (Fig. 24 *c*).

2. When each third beat of the regular ventricular rhythm is replaced by a premature beat, we find a grouping of the apex thrusts in threes, of which the third beat in each group is premature. The arterial beats (α) are grouped in threes,* with groupings of the apical sounds, so that two normal heart sounds alternate with a group of *four* sounds (Fig. 24 *d*); or (β) are paired with grouping of the apical sounds, so that two normal heart sounds alternate with a group of *three* sounds (Fig. 24 *e* and 30).

3. Premature beats which alternate with rhythmic beats give rise to pairing of the apical thrusts (Fig. 27, 28, 32), and to (α) pairing of arterial beats of which the second stroke is weak, and to groupings of heart sounds in fours (Fig. 24 *f* and 29); or to (β) halving of the rate of the arterial pulse, and heart sounds in groups of threes (Fig. 24 *g* and 28).

The differentiation of premature auricular and ventricular beats is not always possible without full instrumental examination.

Where an occasional premature beat occurs, the indications of its *ventricular* origin are as follows: (α) There is no disturbance of the fundamental heart rhythm. The presence of this phenomenon may sometimes be elicited in feeling the radial pulse by anticipating the points at which the rhythmic beats, following the disturbance, ought to fall to carry on the original rhythm; but it is usually more easy to identify instances of disturbance of the rhythm than to exclude them by this method. A strip of radial curve alone is almost always sufficient to distinguish one from the other; in the instance of the premature ventricular beat the full period

* Premature beats may also be responsible for groups of three arterial beats when they replace each fourth rhythmic beat (Fig. 26).

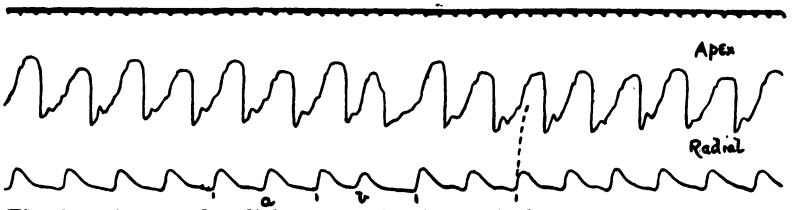

Fig. 25. Apex and radial curves, showing a single premature ventricular contraction. $a = b$.

Fig. 26. Radial curve and heart sounds in a case in which premature ventricular contractions replace each fourth normal beat. The premature beats fail to affect the pulse.

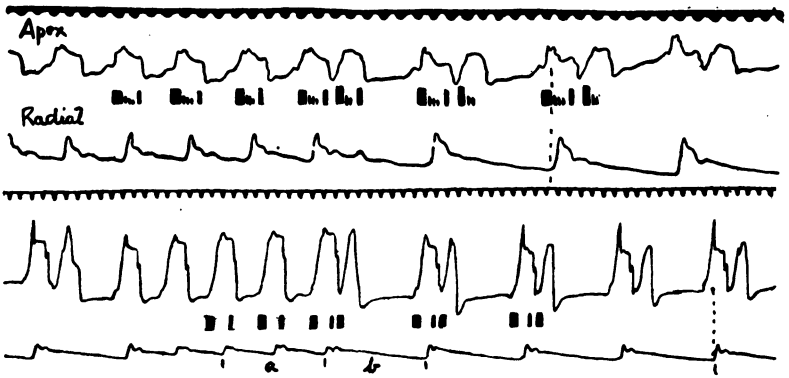

Fig. 27 and 28. Apex and radial curves and heart sounds. The normal mechanism passes into one in which premature ventricular contractions replace alternate normal beats. Fig. 27 is from a case in which mitral regurgitation was present. In Fig. 28, $a = b$.

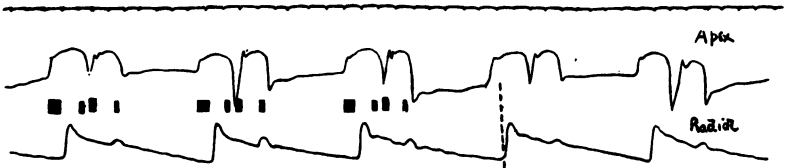

Fig. 29. Premature ventricular contractions replacing each second normal beat. The heart sounds are grouped in fours; the pulse is of the form termed *pulsus bigeminus*.

of the disturbance is equal to two normal cycles (Fig. 25). In the instance of the premature auricular beat the full period is less (Fig. 31). (*b*) There is a prominent jerk and swelling of the veins of the neck (Fig. 30) at the time of occurrence of the premature ventricular beat. This is brought about in the following fashion. The ventricular beat, falling prematurely as it does, usually coincides with a rhythmic auricular contraction, so that the two heart chambers are in systole together (see Fig. 22). As a consequence of this simultaneous contraction, the auricle fails, for a single cycle, to empty itself into the ventricle, and pumps the blood back into the veins. (*c*) By synchronism of the premature ventricular beat with the rhythmic auricular contraction, the corresponding first sound is often exaggerated.

Where the premature beat follows pairs of normal beats or alternates with normal beats, signs *b* and *c* may be present, but *a* is usually valueless unless a transition from a period of disturbance to a period of normal rhythm is graphically recorded (as in Fig. 28 and 32). A comparison can then be instituted between the lengths of the disturbed and undisturbed heart cycles. Thus, in Fig. 28 the long pauses are exactly twice the length of the short ones; *a* is equal to *b*; premature contractions arising in the ventricle have created an exact halving of pulse rate. In Fig. 32 the pause following the premature contraction is not compensatory; *a* is longer than *b* (see Fig. 23).

The effect of premature beats upon the auscultatory signs, when murmurs are present, are manifold; yet most of them can be foretold if the general principles are grasped. A systolic mitral murmur will be found with the premature as well as with the rhythmic beat (Fig. 27), but it is usually short and may be absent. At the base in aortic disease, a systolic or diastolic murmur is present when the premature beat raises the aortic valves (Fig. 33). On the other hand, in mitral stenosis, a presystolic mitral murmur is absent whether the premature beat is auricular or ventricular, but in the former instance, it is often replaced by a presystolic sound. The absence of the presystolic murmur in the case of the auricular beat is attributable either to weakness of the premature contraction or to its coincidence with the preceding ventricular systole.

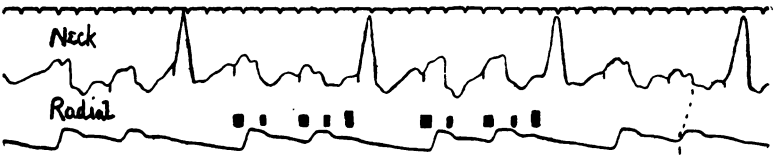

Fig. 30. Curves from the neck and radial artery. Premature ventricular contractions replace each third normal beat, but do not affect the pulse. An exaggerated first sound and a prominent wave, easily visible in the neck, accompanied each premature beat; these phenomena result from simultaneous contraction of auricle and ventricle.

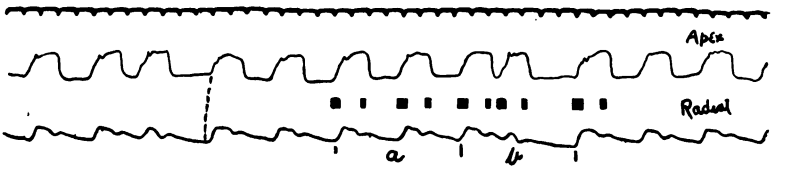

Fig. 31. Apex and radial curves showing occasional premature auricular contractions; *a* is greater than *b*.

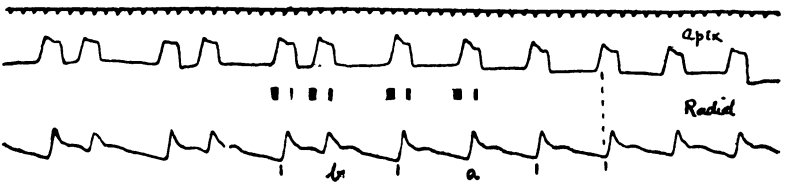

Fig. 32. A " bigeminy " or coupling of heart beats, resulting from premature *auricular* contractions, passing into the normal rhythm; *a* is greater than *b*.

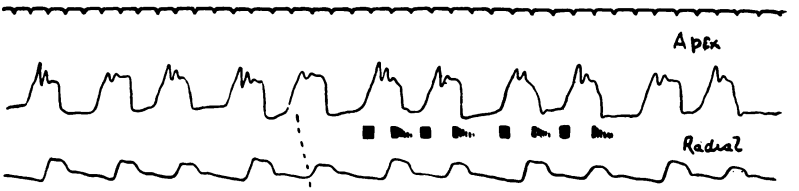

Fig. 33. A bigeminy, resulting from premature auricular contractions. The beats are paired in apical and radial curves. Aortic regurgitation was present.

More complex heart sounds are heard in instances where a premature beat raises the pulmonary, but not the aortic valves, as sometimes happens; the second pulmonary sound occurs, but the second aortic sound is absent. This phenomenon has been erroneously ascribed to hemisystole, the presence of the second sound of the right heart and the absence of that of the left heart being taken as evidences of activity and quiescence of the respective ventricles.

The subjective phenomena which accompany premature contractions.

In a very large number of those affected, the disturbances of heart rhythm pass unnoticed. On the other hand premature beats are not an uncommon cause of what patients term "palpitation." The symptom is more prominent in young subjects, especially those of female sex and those afflicted by nervous irritability. When numerous, they sometimes occasion actual distress; by calling attention to the heart, they often induce worry and anxiety. The sensations experienced are exaggerated by depression of the general health, by fatigue and by emotion. They are often more noticed after the patient retires for the night, after excessive smoking, after a heavy meal, or after exertion.

As a general rule the premature contraction itself passes unperceived; the long pause which follows awakens a sense of uneasiness or oppression in the chest, or a feeling of "void," while the succeeding contraction of the heart is accompanied by consciousness of shock to the chest wall and frequently by a feeling of gripping in the throat. Patients in whom these sensations are vivid sometimes swallow, cough, or inspire as soon as they experience them. When a number of premature beats succeed each other at short intervals, and consciousness of them is marked, anxiety may be profound, and faintness, coldness of the extremities and even sweating may result.

The prognosis and treatment.

It should be clearly understood that, in speaking of the prognostic value of premature beats, I speak of these beats without reference to the conditions with which they are associated. That when frequent and persistent, they often accompany grave affections of the heart will be evident from a study of the tables already given; but this fact does not materially affect the question before us. The associated lesions give prognostic indications of their own; our inquiries are as to whether a heart, which presents no other sign, can be regarded as healthy and as to whether, in the case of an unhealthy heart, the prospect has an added gloom.

It must be admitted that all such beats are decided evidence of a pathological condition and that the pathological process has its seat in the tissues of the heart. The presence of premature contractions is an indication of disturbance of cardiac nutrition, whether temporary or permanent, but it is an aspect that should not be allowed undue prominence. A number of people are temporarily affected by premature beats which do not reappear, while the heart manifests no sign of further damage, either at the time or afterwards. In such instances it is impossible to suppose that the disturbance of the cardiac function has been more than transient or that the nature of it has been serious. Observations and enquiry also teach that they may be present constantly and for long periods, and that those who manifest them may do so from an early to a good old age, such patients never showing any other sign or symptom of cardiac disability. It may be said therefore, that in themselves premature beats cannot be regarded as evidences of serious involvement of the heart muscle, although such involvement is often found in conjunction with them.

The question can be regarded from another standpoint. The premature contractions, when present and frequent, must

inevitably increase the work of the heart, but the amount of the added burden is not easy to ascertain. It is probably not weighty, for where the muscle is evidently compromised and frequent premature contractions occur periodically, little change in the condition of the patient can be detected from time to time, and serious embarrassment of the circulation as a direct result of them is only suspected on rare occasions.

Modern observations therefore tend to minimise the significance of these beats; in fact it has been taught that they may be neglected in the forecast. My own standpoint is a more guarded one. Premature contractions constitute and bear witness to defects; there is the mechanical imperfection and there is the evidence of altered cardiac nutrition; and the more frequent the interruptions, the greater the degree of such defects. Moreover, single premature beats testify to the presence of a process which may lead to cardiac irregularities of a more serious nature. They may be precursors of grave conditions which are considered in subsequent chapters. Premature beats, true paroxysmal tachycardia and fibrillation have a common pathological basis; they are one and all the outcome of new impulse formation in the heart. While it is true that the majority of hearts which show premature contractions may never exhibit profounder derangements, it is also true that these occur for the most part in hearts in which single interruptions have been common; it follows that of hearts seen to-day, some of which show premature contractions and some of which show none, the incidence of grave irregularities will in later years be greater in the former than in the latter.

I may summarise in the statement that, while premature contractions have unquestionably a relatively insignificant import, as compared to many forms of cardiac irregularity, entire neglect of their presence is not advisable. Although

their detection should not be allowed, in itself, to cripple or hamper the patient who is the subject of them, a re-examination of such cases from time to time is recommended.

The first standpoint of treatment is already indicated. The presence of premature beats does not call for a limitation of bodily exercise; it should not prejudice the vocation or pastime of the patient. Restrictions are necessitated only where other signs render them advisable, or where some particular act or occupation is definitely known to originate symptoms of a distressing kind. The anxiety to which the beats conduce in some subjects may be materially allayed by reassurance. No drugs are known which influence their prevalence; digitalis as a direct measure is contra-indicated. The symptoms are usually masked or considerably modified by the bromides administered in doses of from fifteen to thirty grains or more a day; and these drugs are especially useful in tiding a nervous or excitable patient over a period of disturbance.

CHAPTER V.

PAROXYSMAL TACHYCARDIA.

Definition.

Paroxysmal tachycardia is a term which has been and still is applied to several distinct phenomena. It will be of material assistance at present if I use it in a restricted sense and define it as a condition in which from time to time the normal mechanism is abruptly submerged in rapid contractions of the muscle in response to a series of new, rhythmic and pathological impulses.*

The nature of paroyxsmal tachycardia.

It has been stated that the normal pacemaker of the heart lies at the union of the superior cava and right auricle. The usual rate at which the rhythmic impulses proceed from this focus is 72 per minute in the adult. If a new centre of impulse formation developes in any portion of the heart wall, and this centre initiates muscle responses at a rate surpassing that of the normal rhythm, then, while it is active, the new centre dominates the movements of the whole heart. Such are the paroxysms which we are about to study ; they consist of sudden accelerations of heart rate in response to the awakening of new pathological impulses. The paroxysms

* In so defining it I have purposely excluded all accelerations of normal or sinus rhythm, for these are dependent upon innervation. I have also excluded a form of irregular tachycardia which is related to that described in this chapter ; it will be considered in the following chapter.

may be regarded both clinically and pathologically as formed essentially of a regular series of premature beats. The new impulses are elaborated in a single focus, whence the regularity of the series, and this focus lies, usually or always, at a point which is removed from the pacemaker.* Fig. 34 opens with

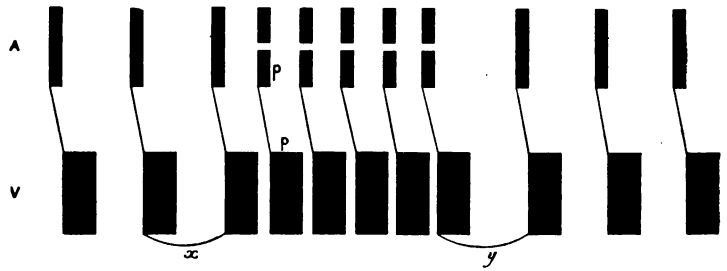

Fig. 34. A diagrammatic representation of a short paroxysm of premature auricular beats; a paroxysm of tachycardia. The abnormal auricular beats are broken in their centres. Each yields a ventricular response. The first abnormal beat occupies the same position in relation to preceding events as does that of Fig. 23. The short paroxysm ends in a pause *y*. *y* is longer than *x*.

three normal heart beats, and the fourth auricular contraction (*p*) is premature. Up to this point the diagram is identical with that shown in Fig. 23†; it differs from the earlier picture in the repetition of the abnormal contraction, five such beats following each other in rapid and regular succession. In each instance the ventricle responds. The paroxysm terminates, and its end is marked by a pause (*y*) which is longer than the pause (*x*) intervening between the beats of the normal rhythm‡; its length is generally that of the pause which succeeds an isolated premature contraction (see Fig. 23).

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* A statement which is based upon the findings of electro-cardiographic curves.

† In both diagrams I have broken the auricular rectangle, to emphasise the abnormal birth place of the pathological contractions.

‡ The interval (*x*) has been deliberately chosen at the onset of the paroxysm, because the restored rhythm of the old pacemaker is often slow for a few cycles. For simplicity this retardation is not figured but it will be referred to again at a later stage.

How important a clear pathological conception of this disturbance is, will be evident: for the nerve control of a new centre of impulse formation is not known from analogy; as a matter of fact the new rhythms show only limited subordination to vagal and sympathetic control.

The sites in which the new rhythms develope are numerous; the abnormal focus is generally seated in the auricle, and the usual sequence of contraction is consequently maintained in the heart chambers; but it may be ventricular, and the auricle then responds inversely to the ventricular beats. The chief features of the mechanism will be sufficiently impressed by a closer examination of the commoner auricular variety. The diagram exhibits a paroxysm of five beats, and this short series permits the display of both onset and offset. Actually the attack may last a few seconds or several months; whatever its length the mechanism is constant, but the symptoms vary with the duration.

The total range of rate in new rhythms is from 110 to 320 per minute; but during most paroxysms the heart contracts 140 to 190 times per minute.

Etiological and pathological relations.

Age. Paroxysmal tachycardia occurs at all ages after the first decade. The actual age limits, so far as observed cases are concerned, are 11-74. In my own series, which comprises 29 cases, the age distribution is as follows:—

Age	10-20	20-30	30-40	40-50	50-60	60-70	70-80
Cases	2	8	4	3	6	4	2

Now these figures are small, for the condition is rare when compared with other cardiac disorders. I have consequently searched for previously recorded cases, and find several reported as early as the 11th year; taking all figures together, the highest incidence is between 30 and 60.

Sex. In my series 18 males and 11 females are included. That is to say, the disorder is a good deal more frequent in men than in women. This proportion is in fair agreement with other records, though perhaps the male element is less predominant in the whole number.

Heredity has been blamed, but the evidence is insufficient to show that it has any direct influence.

Relations to infective disease. In quite half the cases no history of previous illness, other than perhaps children's ailments, can be traced. Rheumatic fever is the only infection which is common. In my own series, nine patients had suffered from it and one had had chorea. Previous records show this history less frequently. Occasional instances appear to have followed immediately upon malaria, measles and scarlet fever; a few of the patients have been syphilitic.

Associated conditions. Most cases of paroxysmal tachycardia are found to have no sign of valve lesion, and in a large number of the patients there is little or no evidence of dilatation during the intervals between the paroxysms.

Nevertheless a number of them show a limitation in the field of cardiac response and become breathless with slight exertion. Taking dilatation, in the absence of valve lesion, undue breathlessness upon exertion and the subsequent development of more serious signs of cardiac failure as evidences of degeneration of the myocardium, I have placed nine of the patients in a corresponding group. The only valve lesion which figures prominently is mitral stenosis, being present in ten of my cases.

Paroxysmal tachycardia and associated conditions.

Mitral stenosis	10
Myocardial degeneration	9
Arterial disease (with and without angina)	2
Aneurism (thoracic)	1
Renal disease and cardiac dilatation	1
Early pulmonary tuberculosis	1
No other signs	5
	29

Factors promoting attacks. Exertion or emotional disturbance are the chief excitants of attacks in those predisposed to them, and the number of instances in which the history tells of paroxysms evoked in these ways is remarkable. The induction of a first attack by unaccustomed effort is often responsible for their hasty assignment to overstrain, but it is questionable if strain is ever the complete story; more probably, damaged or perverted muscle is in all cases the underlying mischief. Flatulence, other digestive disturbances, and especially the assumption of certain postures are amongst the chief remaining excitants of crises.

Morbid anatomy. In the instances in which examination has been possible after death, the most prominent and frequent lesions have been in the walls of the heart. Fibrosis, pallor, friability, atrophy and interferences with the arterial supply are the chief naked eye descriptions. The minute anatomy still remains to be discovered. In a few cases of tachycardia nerve lesions have been found, but their association with the specific condition with which we now deal is more than doubtful.

The recognition of new rhythms.

A heart rate of 180 or more in an adult is usually the result of pathological impulse formation, and especially is this the case where a heart lesion is known to be present. *The rate of the ventricular beating is preserved when the patient passes from the upright to the recumbent position*; it is rarely altered by more than a few beats per minute even when he is maintained in a supine position for considerable periods of time. A physical sign of the utmost diagnostic importance may be noted at the onset or offset of an attack, the increase and decrease in rate at these times is absolutely abrupt. In patients who are conscious of the rapid heart action, but in whom the offset and onset cannot be observed, the sudden

change at the beginning or ending of the attack can usually be elicited by careful questioning.

Other physical signs which may be present are of importance, though their significance is not so great. A prominent and *palpable* pulsation in the veins of the root of the neck is often present. The arterial pulse is frequently irregular in force, and at the first examination may give an erroneous impression of an irregularly beating ventricle. No observations are more unreliable than counts of pulse rates taken in the ordinary manner during the paroxysms ; they should always be checked at the apex beat, either by palpitation or auscultation. The heart sounds are tic tac in character and murmurs which may have been noticed on previous occasions usually disappear while the heart rate is raised. The last sign is of value in mitral stenosis, in which such attacks are relatively common ; for the presystolic murmur is abolished. When a rough presystolic murmur is lost by a patient who develops an accelerated and regular heart action, the disappearance of the murmur is generally attributable to the onset of an abnormal rhythm. In patients who suffer periodically from tachycardia, the presence of occasional premature beats during the periods of quiescence is extremely suggestive that the tachycardia is due to new rhythm production.

The curves are illustrated by Fig. 35-37. In Fig. 35 the onset and offset of the period of tachycardia, due to abnormal impulse formation at a new auricular focus is shown. The slow periods to left and right of it are irregular, for premature contractions interrupt them. The terminations of two long paroxysms are shown in Fig. 36 and 37. The noteworthy features of such curves are several. The changes from the slow to the fast and from the fast to the slow ventricular rates are quite abrupt. Following each paroxysm is a relatively long pause, and this forms the first of a series of

pauses in a period of retarded rate. The rate at the actual termination is almost always slower than the average rate during the periods of quiescence ; quickening, which is best seen in Fig. 36, occurs directly after the termination. The

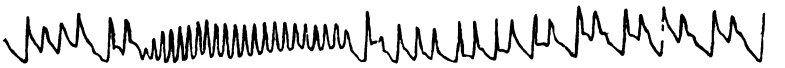

Fig. 35.

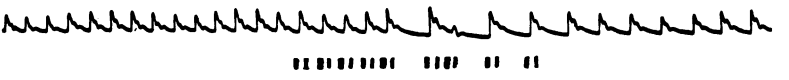

Fig. 36.

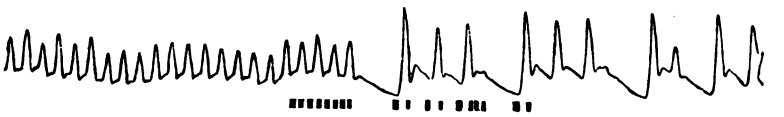

Fig. 37.

Fig. 35 to 37. Three radial curves, taken from separate cases of paroxysmal tachycardia. In Fig. 35 a short and complete paroxysm is shown. In Fig. 36 and 37 the terminations of longer paroxysms are seen. Note the abrupt onset and offsets of the paroxysms, the pauses in which they terminate, the irregularity of the slow periods and the regularity of the fast periods.

slow rhythm is interrupted by occasional premature contractions ; these may usually be shown, by special methods, to have the same point of origin as the paroxysm.

Symptomatology of paroxysmal tachycardia.

Broadly speaking, the less frequent the attacks, the longer do they last. In a given patient, the duration of attacks is fairly constant, so that the paroxysms are similar from time to time. Paroxysms of a few seconds duration are not uncommon; attacks which last for several hours are the most frequent; new rhythms of several weeks or months duration are rare. The attacks may be of any intermediate length.

The symptoms accompanying paroxysms of tachycardia of the kind considered are variable both in their nature and in their degree. They are intimately dependent upon the duration of the attack, the heart's rate during it, and upon the functional response of the heart. Amongst those in whom the attacks are brief, it is not uncommon to find that a patient is entirely oblivious to the rapid heart action when it occurs, and this is more especially the case when the subject is elderly and of the phlegmatic type; or he may be conscious of transitory attacks only when his attention is specifically drawn to them and to phenomena commonly associated with them. Paroxysms lasting half an hour or longer are almost invariably accompanied by obvious symptoms, and these are aggravated as the attack proceeds.

The immediate onset is signalled by a sense of discomfort in the region of the heart, and this discomfort may amount to slight or violent palpitation. A tremor or fluttering in the chest and a beating in the neck are common. General effects, such as lassitude, exhaustion, coldness and sweating are also amongst the early symptoms. Later, flatulence, salivation, nausea and vomiting are prominent. These alimentary symptoms are common within an hour or more of the onset and, once established, usually persist so long as the heart rate is maintained. They hasten the exhaustion which is common and conspicuous in attacks of long duration.

In many patients a number of symptoms which are directly referable to the heart are added. These may be divided into two groups. First, anginal symptoms, varying in intensity from slight precordial pain or a sense of compression with skin tenderness, to violent and continuous pain, radiating in the characteristic fashion over the chest, into the neck, into the left arm or both arms and into the abdomen. Wide areas of hyperalgesia, corresponding to the distribution of the lower cervical and upper thoracic nerve roots, are frequently present and persist after the attack has ceased; they are accompanied by tenderness of the tendons of the sterno-mastoids and of the bellies of the deltoid, pectoral and other muscles. The patients complain of constriction of the chest, variously described as "a band of tightness," "a sensation of gripping" or "a difficulty in breathing." The second group of symptoms is a sequel to embarrassed emptying of the heart. In a number of patients, as the attack proceeds, the limits of cardiac dulness move steadily away from the middle line, and as pallor, which is often an early symptom, becomes more marked, cyanosis and venous general engorgement are added. The veins swell progressively; the eyes seem sunken, dark areas appear below them and the patient becomes restless. The liver bulges downwards, its edge becomes palpable and may pass the umbilicus. Tenderness is experienced when the organ is pressed upon, and pulsation is felt in it; the abdominal muscles assume an increased rigidity; aching pain developes in the epigastrium and right hypochondrium. In more exceptional cases, puffiness of the ankles and face develope after a long continued attack. The spleen may also show signs of enlargement. A cough, accompanied by a frothy and sometimes blood stained sputum is not infrequent, and signs of engorgement of the lungs in the form of sibilant rhonchi and moist râles are found at the bases. Collapse of the patient is prominent in the later stages. The attack may terminate

in progressive failure, delirium, ascites, general anasarca and death. Unexpected death also ends the attack on occasion, but the great majority of the paroxysms cease at the abrupt resumption of the normal rhythm. The actual cessation of the attack is marked by symptoms of its own, a sharp stabbing pain in the chest, or one or more forcible thumps of the heart. But as a rule the patient speaks only of relief. Nothing is more remarkable than the rapidity with which the natural circulatory conditions are restored, when the abrupt fall of pulse rate comes. The dilatation of the heart and the accompanying engorgement of the neck veins vanish as it were by magic. The liver recedes beneath the ribs, respiration becomes free, the pain is subdued and the remaining symptoms subside. Quantities of flatus and limpid urine are often passed after an attack.

A varying degree of exhaustion follows the severe attack, the cough may continue for a few hours or days, and skin and muscle tenderness commonly persists for some while.

Differential diagnosis.

The diagnosis of paroxysmal tachycardia, during an attack, rests upon careful attention to the history of the patient and to those physical signs and symptoms which have been enumerated already. As a rule there is little difficulty. But a number of errors do occur, and the chief of these may be mentioned; they mostly depend upon the prominence of symptoms which are referred to other organs, and consequently upon a hurried or neglected examination of the organ at fault.

The stasis of the lungs, with dulness and crepitations at the bases has been attributed to pneumonia. It is an error which should not happen, for it is always accompanied by signs of venous congestion in other organs. When it has occurred, I believe it has been largely attributable to under

estimation of the heart rate, and the mistake emphasises the rule that the heart rate should be taken from the apex beat and not from the wrist.

Anginal pain, maximal in the abdomen and accompanied by abdominal rigidity, vomiting and signs of collapse has been mistaken for the symptom of a perforated gastric ulcer, and has led to a dangerous and needless laparotomy; and this in a patient in whom cardiac dilatation, engorgement of the veins and excessive heart acceleration, were overlooked in the absence of conspicuous cyanosis.

A large number of cases are grouped under the comprehensive term "heart strain," and this is applied especially to the patient in whom the first attack has been hastened by effort.

More than one instance has come to my notice, in which "acute cardiac dilatation" has sufficed as a diagnosis in a pregnant woman, suffering in reality from a rheumatic heart with mitral stenosis. A rheumatic history is not uncommon in cases of paroxysmal tachycardia, and the characteristic murmurs of mitral stenosis, when this valve lesion is present, are usually masked during the attack. A history of rheumatic fever, or a slight systolic thrill and an accompanying apical murmur, may suggest a more correct interpretation of the case.

The chief difficulty arises, as these instances illustrate, when a patient is seen for the first time in an attack, and this is especially so when no clear history is obtainable. When a regular heart rate exceeds 160 per minute in an adult, the presence of a new rhythm, rather than acceleration of the normal rhythm, should always come first to mind; it may be suspected even at lower rates. The reaction of the rate to posture is important. It is perfectly true that very high pulse rates are met with in exophthalmic goitre, in pulmonary tuberculosis, in alcoholism and other conditions, but the

presence or absence of the diseases or intoxication in question may usually be ascertained and the conditions differentiated. Failing positive evidence from these sources, an examination of the heart rate in its response to posture is of service. In the aforesaid conditions a notable or marked decrease of rate at or shortly after the assumption of the supine posture is the rule. Where we deal with a new rhythm, posture influences the rate inappreciably, if at all, neither is it affected by repeated swallowing or the suspension of respiration. A persistent tachycardia of 140 or upwards, maintained under a variety of circumstances, should always suggest the presence of a new and extraneous heart rhythm.

Patients who are the subjects of relatively brief attacks occasionally seek advice during periods of quiescence on the score of attacks of faintness, palpitation, rapid heart action, etc.. The true nature of the condition may be suspected or proved by careful examination. The history of the sensations at onset or offset are then most valuable. The complete absence of symptoms or physical signs of cardiac involvement, and especially the absence of occasional or frequent premature beats, should suggest causes other than those which we are considering, though they are not finally excluded thereby. In a neurotic subject, excessive force of the cardiac action and excessive consciousness of the beat are the most probable explanations. In cases of doubt an effort should be made to investigate the heart during an attack. A prolonged examination of the patient is sometimes rewarded by the discovery of brief paroxysms of true paroxysmal tachycardia, for the patient so affected is often the subject of more attacks than those of which he is conscious.

The prognosis.

The prognosis of the individual attacks contains an element of uncertainty. Death during paroxysms has

occurred on not a few occasions, but the great majority of the paroxysms are recovered from. Several prognostic aspects need emphasis. The symptoms of the patient are largely governed by the reaction of the nervous system; neurotic subjects, especially women, awaken needless anxiety. The duration of the observed paroxysm and the length of previous seizures have to be considered. The outlook is more ominous when, after a continuation of several days, the heart shows signs of progressive weakening, manifested by steady increase in its size and by the supervention of lung and abdominal stasis. The strength of the pulse is no indication of the future, it may be scarcely perceptible in repeated attacks. The gravest symptoms are those of increasing respiratory embarrassment, consequent upon oedema of the lungs, and the onset of delirium and general anasarca. Nevertheless it often happens that when embarrassment is profound the paroxysm ends, and the patient passes in a few minutes from a condition of acute distress and seemingly the utmost gravity to one of relative comfort and safety.

The prognosis of the malady as a whole should be based upon two chief considerations; first and most important, upon an estimate of the endurance of the cardiac muscle, and secondly upon the severity of the trials through which it passes. The estimate of the first factor is formed from the signs and symptoms between the attacks and from the reaction to moderate effort. The prognosis in a case of paroxysmal tachycardia is the same as that in a similar case which shows no attacks, but with the following reservations: the attacks are themselves important indications of muscle damage, and the attacks frequently place the life of the patient in jeopardy. The reaction of the heart to the attacks is also of importance. A healthy heart reacts to a pure increase of rate, amounting to a doubling of the normal rate, by *decreasing* in size, and the circulation may be maintained

for long periods. A diseased muscle reacts by dilating. The degree of dilatation and the rapidity of its onset and progress consequently suggest the degree of muscular involvement.

The estimate of the second factor involves a survey of the length and frequency of the attacks and the heart rate during such attacks, as they are summed up by observation and the previous history; but as the attacks may cease at any time never to return, and as we are ignorant of the grade of injury, if such injury exists, which single paroxysms impose upon the heart, the value of these considerations in the completion of the prognosis has its limitations. The possibility of death in a seizure is an uncertain factor; it necessitates caution in prognosis when the paroxysms are of long duration.

The prognosis, where little further sign of cardiac muscle damage is found, and where the paroxysms are infrequent and of a few hours duration, and the rate not very excessive, is favourable; such paroxysms do not curtail life as a rule, and a prospect of long years may be spoken of to young subjects without hesitation. These patients always wish to know whether they will ever be free from attacks. They may be told that, although such freedom cannot be promised, the prospect of it is fair. The prognosis as a whole starts from this foundation, and as muscle or valve lesions are more in evidence, as the attacks are longer and more frequent, as the heart acceleration is greater, and as the patient is older, so the outlook is less hopeful.

The treatment.

The treatment of paroxysmal tachycardia may be conveniently dealt with from two standpoints; the management of the attacks themselves, and the care of the patient during the general course of the malady.

Are we aware of any remedy which will infallibly abolish a paroxysm of tachycardia? The answer to this question is in the negative. I have frequently seen attacks of several hours duration terminate shortly after the administration of certain remedies or after certain interferences. The patients who are the subjects of them are frequently aware of and adopt certain curative measures. In some instances, the assumption of a given posture, sitting and placing the head between the knees for example or lying supine, is a certain remedy. The induction of vomiting, the relief of flatulence, firm pressure upon the carotid sheath and its contained vagus nerve on one side, the application of a tight abdominal binder may be immediately and constantly efficacious in given cases. I have seen the application of an icebag to the precordium, a remedy which always affords relief, speedily terminate attacks. Similarly they have ceased shortly after the administration of a single intravenous injection of digitalin (1-100 gr.) or strophanthin (1-250 gr.). But much more often than not, such remedies are absolutely without effect and the treatment finally adopted becomes palliative or symptomatic. Rest is enjoined, and attention is paid to the wishes of the patient in respect of posture. Most frequently these unfortunate people prefer to lie well propped with pillows; sometimes they prefer to stand. The dietary should be fluid, bland and as restricted as possible. Iced water or milk are well borne and are often beneficial.

Local applications, the icebag, a mustard plaster, leeches or cupping, over a distended or pain-giving organ, be it the heart or the liver, often afford great relief. Pain, if general, may be combated by more general remedies, such as chloral or morphia; but these drugs are not often called for. The induction of sleep in long continued paroxysms is essential, and fortunately chloral and the opiates may be employed with safety. Serious engorgement of the hear-

and signs of progressive lung oedema or grave venous stasis are indications for venesection. The letting of 8 or 12 oz. of blood will be followed by improvement; but the occasion does not often arise. Respiratory embarrassment is relieved and sleep induced by the administration of oxygen; this gas is best given through a light fitting mask which covers the whole face, so that high percentages are breathed.

The treatment of the malady as a whole is largely governed by the condition of the heart between the attacks. A searching inquiry may reveal exciting causes of paroxysms; often, sudden exertion or emotion is the chief provocative, so that the cessation of employment becomes, not infrequently, imperative. General care of the health, the cleanliness of the mouth and throat, the orderliness of the dietary and the remedying of dyspeptic troubles and constipation may ward off the crises. The continued wearing of an abdominal belt, applied before rising and omitted at bedtime is sometimes accompanied by the happiest of results.

Where other remedies fail, a full course of digitalis* may ultimately improve the condition.

The paroxysms themselves do not contra-indicate the careful administration of general anæsthetics, should they be necessary.

* By a full course, I wish to denote a course of the drug which will produce a definite reaction in the form of nausea or headache, and the subsequent administration of the drug for several weeks, in doses which are tolerated. As a rule ʒss-ʒi of the tincture or ʒss-ʒi of the fresh infusion may be given daily for the first week, the dose being increased until symptoms appear, and finally reduced to the maximal quantity tolerated without undue discomfort. Small doses of digitalis and the allied drugs are without appreciable effect. Aconite, strychnine, belladonna and its allies should be avoided.

CHAPTER VI.

AURICULAR FIBRILLATION.

Definition.

A condition in which normal impulse formation in the auricle is replaced by stimulus production at multiple auricular foci. Co-ordinate contraction in the auricle is lost; the normal and regular impulses transmitted to the ventricle are absent, while rapid and hap-hazard impulses produced in the auricle take their place and produce gross irregularity of the ventricular action.

The nature of auricular fibrillation.

When we inspect the normally beating heart of an animal, the systoles of both auricle and ventricle are readily discerned. The movement of the auricle is a sharp flick, most clearly perceptible in the length of the auricular appendix, for in this line the shortening is greatest. When the auricle is caused to pass into fibrillation or delirium, the appearances are quite distinctive; the muscular walls are maintained in a position of diastole; systole, either complete or partial, is never accomplished; the structure as a whole rests immobile; but close observation of the muscle surface reveals its extreme and incessant activity, rapid and minute twitchings and undulatory movements are visible over the whole. It is believed that the tissue mass has suffered functional fragmentation and that a number of small areas

give independent birth to new impulses. Further it is held that these fresh impulses are pathological, being similar to, or identical with, those which evoke single premature contractions. The effect of the auricular confusion upon the ventricle is twofold. The normal, regular and co-ordinate contractions in the auricle are in abeyance and consequently the ventricle is robbed of the regular impulses which form its accustomed supply. These are replaced by numerous and hap-hazard impulses, escaping to the ventricle from the turmoil which prevails in the upper chamber; the change in the action of the ventricle, when the auricle fibrillates, is consequently profound. Its rate of beating rises considerably

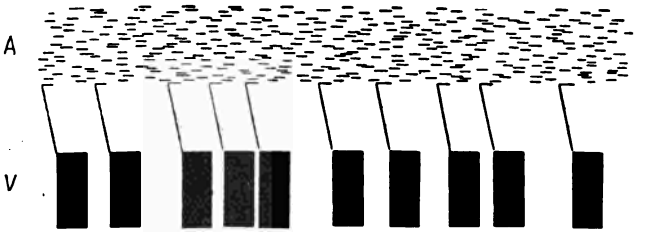

Fig. 38. A diagrammatic representation of auricular fibrillation. The fibres of the auricles do not contract co-ordinately or together, but the tissue is broken up into a number of independently active areas. Occasional impulses leave the auricle at quite irregular intervals and stimulate the ventricle, producing in it a rapid and irregular action.

and the contractions follow each other in a completely irregular fashion.* This mechanism is diagrammatically represented in Fig. 38, in which I have attempted to emphasise the absence of co-ordinate auricular beats, the presence of constant fibrillary contraction, and the irregular responses of the quickened ventricle to the new auricular impulses.

Such are the events in experiment, and those of the clinical condition are identical, with one proviso; since in the

* Ventricular fibrillation is incompatible with life. It is probably responsible for many instances of unexpected death.

experimental heart the tissues controlling the conduction of impulses are healthy, the rate of the ventricular contractions is doubled or even trebled; but in the human subject, the conducting tissues may be either intact or damaged, consequently the ventricular rate varies widely in different patients, according as access to the ventricle is full or limited. While a free passage yields rates approaching 200 per minute, damage to the junctional tissues may reduce the rate to 40 or less: the usual rates lie between 90 and 140.

Etiological and pathological relations.

Age. The observed age limits of fibrillation are 13 to 84. The condition has not been found in the first decade; if it occurs, it is quite an exceptional event. In studying the age distribution, the cases are conveniently divided into rheumatic and non-rheumatic groups. This division clearly shows that, independent of rheumatism, the affection is related to advancing years; as with premature contractions, the highest incidence is in the sixth and seventh decade. In the rheumatic group, the incidence is heaviest between the twentieth and thirtieth years; it is almost as heavy in the fourth and fifth decade, but lightens as the years mount further.

Age distribution of auricular fibrillation in 116 *cases.*

Age	0-10	10-20	20-30	30-40	40-50	50-60	60-70	70-80	80-90	90-100
Rheumatic group	0	4	24	19	21	10	4	0	0	
Non-rheumatic	0	0	0	2	6	13	11	0	2	
Combined	0	4	24	21	27	23	15	0	2	

Sex. Auricular fibrillation is much more common in men than in women, and the preponderance in males is chiefly in the non-rheumatic group. Where there is a rheumatic history, the sexes bear the burden more equally. The relative frequency of rheumatic fibrillation in women is linked with the prevalence of mitral disease in this sex;

mitral stenosis and auricular fibrillation are bosom companions. Among 127 subjects the sex distribution was as follows :—

	Male.	*Female.*
Rheumatic ..	46	41
Non-rheumatic	34	6
Combined ..	80	47

Relation to infections ; associated conditions. Amongst 126 cases, a rheumatic or choreic history has been found 60 times ; in four instances at least, there was a history of one or other affection in the family. Amongst the remainder, mitral stenosis was present in 22, pericardial adhesions or effusion in two cases and subacute streptococcic endocarditis in one case. If these patients are collected to form a rheumatic group, the subdivision includes 89 cases, or 70 per cent.. The prevalence of fibrillation amongst those who suffer from mitral constriction is especially noteworthy ; 66 of the cases, or 52 per cent., had this valve lesion. The relation to mitral stenosis may be traced in another and equally emphatic manner. Of 106 cases of mitral stenosis collected in an out-patient department, 22, or approximately one-fifth, showed auricular fibrillation. The proportion amongst in-patients is much higher ; it exceeds 50 per cent..

In the table, I have classed a group as myocardial degeneration ; it includes those in whom the heart irregularity was the outstanding feature, though many of the cases gave signs of cardiac failure apart from the irregularity. Aortic disease, arterial disease and granular kidney are the most prominent lesions in other groups. Of all cases of cardiac failure admitted to a general hospital 60 to 70 per cent. manifest this disorder of the cardiac mechanism ; it is difficult, therefore, to over-emphasise its importance.

		Mitral stenosis	Myocardial degeneration	Aortic disease	Arterial disease	Granular kidney	Pericardial adhesions and effusion	Remainder	
Rheumatic or choreic history	Mitral stenosis	*42							60
	Myocardial degeneration		10				1		
	Pericardial adhesions			5					
	Aortic disease					2			
	Granular kidney								
Rheumatism or chorea in family	Mitral stenosis	2							4
	Myocardial degeneration		2						
No history of rheumatism or chorea	Mitral stenosis	16							56
	Arterial disease				7				
	Myocardial degeneration		14						
	Granular kidney					4			
	Aortic disease			3					
	Aneurism							2	
	Emphysema & Bronchitis							2	
	Strepto. Endocarditis							1	
	Pericardial adhesions and pericardial effusion						2		
	Tuberculous pleurisy							1	
	Syphilitic heart							1	
	Congenital heart							1	
	Chronic alcoholism							1	
	Pneumonia							1	
Rheumatism or chorea not noted	Mitral stenosis	6							6
		66	26	8	7	6	3	10	126

Of etiological factors, rheumatism is predominant, as we have seen; a history of other infections, "influenza" amongst them, is given by many patients, but the influence of these infections is imperfectly understood.

* The heavy figures mark the rheumatic group.

Morbid anatomy. That valve lesions are present in a number of the cases is obvious from the bedside examinations ; enlargement of the whole heart is common, and dilatation or hypertrophy of the auricles is more frequent than the valve lesions which might be held to account for them. The most constant structural alterations, which are found, are discovered by histological examination of the heart musculature. Usually, it shows a more or less intense grade of subacute or chronic inflammatory change progressing to fibrosis, and the auricles are conspicuously affected. A diffuse fibrosis, accompanied by leucocytic infiltration and atrophy of the neighbouring muscle cells, is the most frequent change.

Such is the tale told by the microscope, but it does not justify us in holding that the inflammatory reaction is the cause of the altered mechanism. We examine the hearts of those who die, and most die with all the classical signs of heart failure. Many of the microscopic lesions are to be regarded as the result of infections producing heart failure rather than fibrillation. Similar lesions are found where fibrillation has never occurred ; and hearts which have shown this disorder may not present the lesions described.

The recognition of auricular fibrillation.

Auricular fibrillation gives rise in a clinical case to two series of phenomena ; the one dependent upon the virtual paralysis of the auricle ; the other dependent upon the irregularity of the ventricle.

It will be convenient to study the ventricular signs first. The irregularity is most varied in form according to the rate of the contractions. When the heart is beating rapidly at 100-160 per minute, the grade of disorder is maximal. The radial artery supplies indifferent news of the ventricular rate, many pulsations fail to reach it (such beats are marked with asterisks in Fig. 40). The pulse is a medley

Fig. 39 to 44. Apex and radial curves from cases presenting auricular fibrillation.

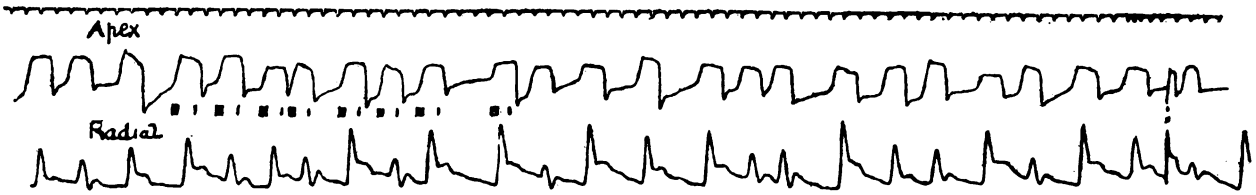

Fig. 39. The heart's action is grossly irregular, each ventricular beat reaches the wrist. The heart sounds are clear but irregularly placed.

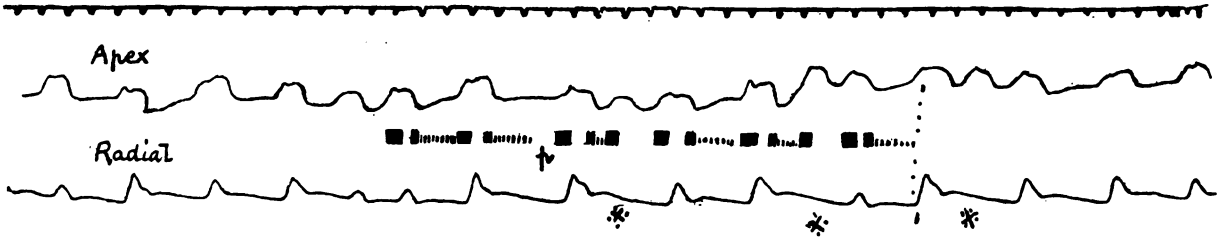

Fig. 40. From a case of mitral stenosis. The heart's action is grossly irregular, many beats fail to reach the wrist (*). A diastolic murmur fills the shorter pauses, but falls short of the first sound in the longer pauses (*p*). The murmur does not constantly occupy the presystolic period.

Fig. 41. Gross irregularity is present. Mitral systolic and aortic diastolic murmurs accompany each cycle.

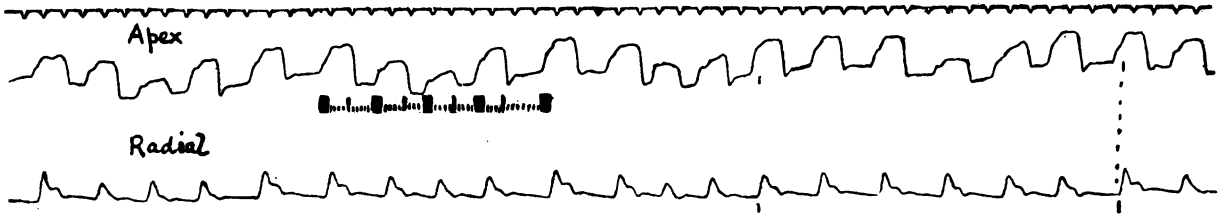

Fig. 42. From a case of mitral stenosis. Mitral systolic and diastolic murmurs completely fill the gaps between the heart sounds.

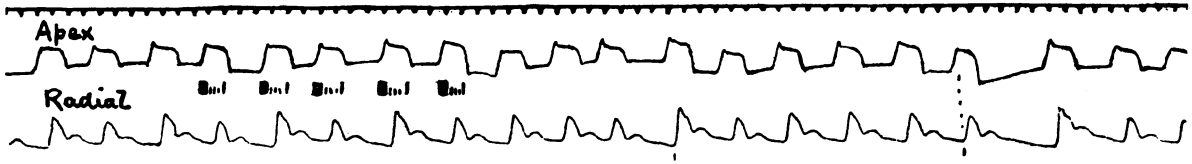

Fig. 43. Showing a lesser grade of irregularity; a mitral systolic murmur accompanies each ventricular contraction.

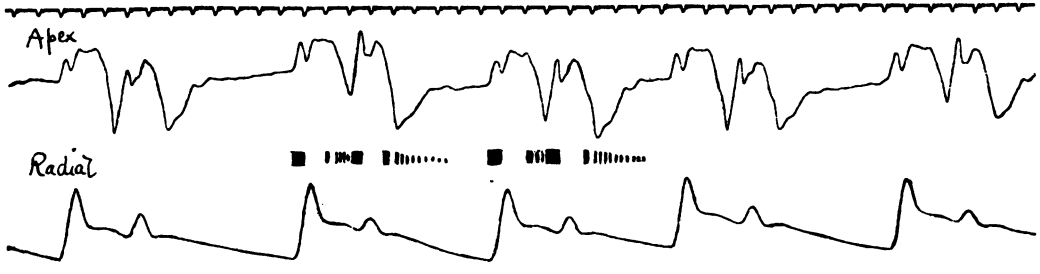

Fig. 44. From a case of mitral stenosis under the full influence of digitalis. "Digitalis coupling" is present. The presence of fibrillation is recognised by the variation in the lengths of the pauses following the weaker pulse beats. The heart's action is slow. A diastolic murmur, commencing soon after but not immediately with the second sound, runs through early and mid-diastole.

of beats of many sizes (Fig. 39), an intimate mingling of changing pauses; now the beats are almost uniform in strength and spacing; now feeble pulsations chase along rapidly; now the pulse is lost; now it returns with increased vigour. Feel the pulse and the mechanism is apparent: the more the disorder, the more certain the evidence. It is when the rate is slow that close attention and more experience are often asked, for with slower rate, the disorder is less pronounced; all the heart beats now reach the wrist and the irregularity comprises minor variations in the length of pause (as in Fig. 42); in such instances it escapes notice, and a heedful examination, concentrated upon its presence or absence, alone brings it to discovery. Short Dudgeon curves reveal the irregularity on all but rare occasions. The nature of the arrhythmia is such that the heart action is never quite regular, and seldom do two beats of a precisely equal character or length lie adjacent. No two whole strips of curve are similar; the pauses betwixt beats bear no simple length relation one to another. Proportion between the force of an arterial pulse and the pause preceding it is often lost (Fig. 39 and 40); a strong beat succeeds a brief pause and a weak beat succeeds a long one. When the pulse is slow, only beat to beat measurement may disclose the irregularity.

The fully developed disorder of the ventricle is readily appreciated at the apex. The heart sounds are modified; they vary in intensity and the variation runs hand in hand with the strength of the beats. First and second sounds are present with each cycle which gives arterial pulsation (Fig. 39); a first sound stands isolated when the pulse beat is missing (Fig. 40). If a systolic mitral murmur is present, it accompanies each ventricular contraction (Fig. 43), except where the rate is fast, for here it is apt to vanish. Aortic murmurs obey the general rule, their presence or absence is controlled by the efficiency of the respective beats (Fig. 41).

The inactivity of the auricle is responsible for special alterations of the heart sounds in patients who have stenosis of the mitral orifice. It is customary to allude to disappearance of presystolic murmurs when the auricle fibrillates, but this statement is not exact. The change in the characters of the murmurs at the onset of fibrillation is oftentimes impressive, but it varies according to the heart rate and the degree of stenosis. If, while the regular heart beats are present, there are short presystolic murmurs, these murmurs usually vanish when fibrillation begins ; and more especially if the rate during fibrillation is rapid. If the presystolic murmur is long and rough, a murmur of similar character is preserved during fibrillation. But its time relations are altered. Attention should concentrate upon the position of the second sound at the apex. When the auricle is fibrillating, the diastolic murmur has a fixed time relation to this sound. If the heart rate is rapid, the murmur begins early in diastole and fills the whole gap to the first sound of the succeeding beat (Fig. 42) ; if the heart rate is less rapid, the murmur maintains its relation to the second sound but falls short of the succeeding first during the longest ventricular pauses (Fig. 40*p*) ; if the heart rate is slow, a long, though varying, interval separates the end of the murmur and the next first sound ; the murmur is then confined to early diastole (Fig. 44). The whole series of murmur arrangements may be observed in a single case which exhibits different heart rates from time to time. The reason of the changes will be clear when the pressures and mechanism are considered. The diastolic murmurs of mitral stenosis are dependent upon the rate of flow through the constricted orifice, and the rate of flow is controlled by the difference of pressure in auricle and ventricle at any given moment. Now although the auricular pressure exceeds the ventricular during the whole of diastole, the excess is greatest at two phases, namely, when the

auricle is in contraction and directly after the opening of the mitral valves. Where the auricle contracts in normal fashion, mitral diastolic murmurs are in chief evidence at first in late, and afterwards in early diastole ; they are found in early diastole when the auricle is virtually paralysed, especially when, the heart rate being slow, stasis raises the ventricular pressure during the last phases of diastole.

The clinical recognition of auricular fibrillation rests primarily upon the nature of the ventricular action, but it is aided, as we have seen, by certain additional phenomena. It is possible, too, to formulate a few general rules, which serve as useful guides to its identification. When the ventricle beats irregularly at a rate surpassing 120 per minute, the irregularity is almost always of this nature. When an irregular ventricular action accompanies signs and symptoms of serious heart failure, it is probably the result of auricular delirium, and the probability is increased if the heart rate is much accelerated. In patients in whom the heart is irregular, but in whom the heart rate is not much accelerated and in whom signs of heart failure are absent or few, a test may be applied which is of considerable value. Moderate exercise augments the ventricular rate, and this is so whether fibrillation is present or not ; but there is a striking contrast in two given cases of irregularity, of which one is due to auricular fibrillation, while the other has a different cause (*i.e.* premature beats, partial heart-block, etc.). In fibrillation the pulse becomes more irregular with its acceleration, while in the remainder the pulse steadies. When premature beats are present, a sufficient acceleration of ventricular rate to abolish them temporarily may often be induced by several quick changes from the recumbent to the sitting posture ; this is not so where fibrillation is concerned. On the other hand, as the pulse slows subsequent to exercise, reversed relations are witnessed ;

the irregularity of fibrillation decreases, while other forms of irregularity become more prominent. Fever similarly raises the ventricular rate and during the febrile stage the disorder of fibrillation persists and is often augmented in degree. Finally, the persistence of the irregularity, which is due to fibrillation, needs emphasis. In most cases it is continuous from the time of observation until death. The other irregularities are present from time to time, so that there are intervals of regular ventricular action each hour or each day.

The general symptomatology.

The symptoms complained of by patients in whom the auricles are in a state of fibrillation are very various, being dependent mainly upon the concomitant conditions. They are the symptoms of degenerate and failing heart muscle, and these do not require reiteration at the present time. The symptoms which are now our special concern are those which appear to be the special developments of fibrillation itself. Patients, who possess the persistent disorder, often experience occasional fluttering in the chest and neck and may be conscious of irregular heart action. They are more prone to shortness of breath, exhaustion and other symptoms of over-taxation of the heart than are those with similar valve lesions and a like degree of cardiac dilatation*; but it is not always easy to allot these superadded symptoms to precise causes; they are in part the result of the graver myocardial condition which consorts with fibrillation; they are in part due to the actual turbulence and embarrassment of the ventricle.† That the heart is taxed by the irregularity cannot be doubted, but it cannot be stated that any symptom,

* On the other hand they seem peculiarly exempt from angina.

† The heart embarrassment is the result of ventricular irregularity; the virtual paralysis of the auricle is without appreciable effect on the general circulation.

such as cyanosis, conspicuous dyspnoea, noticeable venous engorgement or dropsy, is the direct outcome of fibrillation; for cardiac failure and these, its classical accompaniments, are found where there is no fibrillation, and instances of fibrillation are not rare in which these symptoms are not discoverable. In the production of the symptoms, there is, as has been stated, an interplay of two factors, namely, the inherent muscle defect and the extra burden of disordered action; while the signs of failure are proportioned to the degree of muscle damage, the whole of this symptomatic scale is raised by irregularity. In the healthy hearts of animals it is a general rule that fibrillation of the auricle produces a fall of arterial and a slight rise of venous pressure, but at the same time, it is accompanied by a *decrease* in the heart's dimensions, a usual phenomenon when the rate is increased. The heart accommodates itself to the new conditions in a few moments; the arterial pressure rises and the venous pressure falls, so that they almost recover their previous levels and the blood flow is maintained in a well-nigh perfect fashion for hours. But if the heart has been damaged, the effect is both profound and lasting and in place of *decrease* of heart volume, an *increase* may occur. So it is in patients. Patients may experience paroxysmal fibrillation at intervals of a month or perhaps a year; many of them pass through their attacks with little or no sensibility of them; neither can any sign, other than irregularity, be discovered during their progress. Yet similar crises give rise in other patients to profound and serious disturbance, breathlessness, pain, cyanosis and further indications of increasing dilatation of the heart. In these, the severest cases, the symptoms resemble those of long continued paroxysms of regular tachycardia. Between the mild and most extreme reactions is the intermediate. The variation in the reaction is great, and as I have said is largely attributable

to the grade of underlying heart mischief. But there is another and equally important factor in the human subject; it is the rate of the ventricular action during the attack. Just as the muscle defect varies in its degree, so also does the burden imposed upon it; thus, in the extreme instances, it is found that little reaction is shown in paroxysms of relatively slow ventricular action, while amongst those with grave disturbance the ventricular rate is usually rapid.

Remarks upon diagnosis.

The diagnosis, usually suggested for cases which exhibit fibrillation of the auricles, is still that of the accompanying valve lesion, though I am strongly of opinion that it is no longer warrantable. A diagnosis should include either the outstanding feature of the pathology, or it should be chosen that it may become associated with some specially beneficial form of treatment. In all these patients chronic affection of the myocardium is the essential lesion; while the relations of the cardiac disorder to digitalis medication are so peculiar that the named disorder of the heart always brings this drug to mind.

But I wish to refer but briefly to this question of terminology under the present heading; and have chosen it more especially to emphasise a common and avoidable diagnostic blunder, which is the outcome of want of true appreciation of the mechanism in these cases. In discussing the signs associated with fibrillation, I have spoken of the modification of diastolic murmurs in mitral disease. A murmur, which originally occupies the full diastole of the shorter cycles, is replaced, as the heart slows, by an early diastolic murmur which is maximal in the region of the apex. It is the last murmur which so frequently misleads the physician and suggests to him an insufficiency of the aortic valves. It is known that in some cases of aortic regurgitation the characteristic

bruit is *contined* to the apex, but I believe this is far less common than has been supposed, and that an erroneous conception of its frequency has arisen from inclusion of many of the cases to which I now refer. When mitral stenosis and auricular fibrillation are present in the same patient, *and the heart rate is slow*, an early diastolic murmur, most clearly audible at the apex but often spreading beyond it, is an expected sign. A diagnosis of aortic reflux is never justifiable when the heart is grossly irregular and slow, unless unequivocal signs of it are present apart from such a murmur. Uncomplicated aortic valvular disease and fibrillation of the auricles is a comparatively rare clinical picture. The combination, yielding a purely apical murmur, is so far an undescribed condition. Close attention to the character and accurate timing of the adventitious sound is often helpful. The early diastolic murmur of mitral stenosis is relatively soft in quality and it usually begins a little later than the second sound. The absence of a waterhammer pulse and of a murmur at the aortic cartilage are evident aids to a correct conclusion.

The prognosis.

As in all other kinds of heart irregularity, the prognosis is largely governed by the remaining symptoms and signs, and in any individual case an estimate is formed, which includes consideration of the past history, the presence or absence of serious symptoms, the presence or absence of dilatation, of valve lesion, renal disease, etc.. But fibrillation gives an added significance to the case. It is, as I have said, in itself an evidence of muscular damage, and of serious muscle damage. It loads an already defective muscle with an extra and appreciable burden. In most cases it heralds cardiac failure, temporary or terminal, so that few patients survive its onset for more than ten years. There are well

authenticated instances in which it has persisted for a longer period, but they are few. The most valuable intrinsic sign is the rate of the ventricular action, a persistent rate of 120 or over is of serious omen, and according as the rate is maintained above this count, so the outlook becomes graver. Rates of 140 and over are rarely maintained for many months, rates of 160 do not continue for many weeks. An extremely important consideration is the reaction to treatment. As we shall see, a large number of cases react to cardiac drugs; in many, more especially the rheumatic group, the rate can be controlled, can be reduced and can be maintained within limits which spare the heart from excessive taxation of its strength. In dealing with a patient who has a given heart rate, the prognosis, in so far as it is affected by the fibrillation, does not depend so much upon the rate of the heart beat observed, as upon the heart rate which persists under treatment. At the same time, the prognosis is more grave for a given rate, when treatment is required, than when this rate is maintained in the absence of remedies.

The treatment.

There is no ailment in which such success can be achieved, no other cardiac disease which may be so speedily benefited, as the well-managed case of auricular fibrillation. In no other affection can the medical attendant point with more thorough confidence to the effects of his remedies. As a direct result of active treatment the moribund may be restored and many years may be added to their lives. Auricular fibrillation is the condition to which drugs of the digitalis group owe their well-founded reputation.

The guide to the physician is the rate of the heart beat, an index which rarely fails him. Auricular fibrillation is an absolute indication for the administration of a member of the

digitalis group, whenever the heart rate exceeds 100 while the patient is at rest. In a very large proportion of the patients the drug acts as a specific, impeding the passage of impulses from auricle to ventricle and thus reducing the rate. If the heart rate does not fall as a result of rest, and if it will not fall when digitalis or an allied drug has been properly administered, no other remedy is known which is of service in reducing the heart rate. In young people, and especially those who have been affected by rheumatism or chorea, an absolute control of the rate is almost always established and maintained. The treatment consequently consists of the administration of such doses as will keep the heart rate within reasonable limits.

It does not necessarily follow that a patient who has fibrillation should lie up. But where the heart rate exceeds 100 it is advisable, and the patient should remain in bed until his reaction to digitalis, or a similar drug, has been thoroughly investigated. Further treatment in bed is decided upon according to the general condition, and according to the tolerance and reaction to digitalis. In treating cases with digitalis, it is found that in some the rate is unaffected; these are chiefly patients of the non-rheumatic group. In most, a reaction is speedily obtained. These latter may be divided into three classes: the first, those in whom the reaction is a permanent one; these are patients in whom the rate remains slow though digitalis is omitted: the second, those in whom the reaction is permanent when small doses are subsequently administered: the third, those in whom persistent high dosage is required to maintain control.

As a routine, the tincture or fresh infusion of digitalis is given, for it is the safest and most potent remedy. The tincture is given to adults in doses of from 10 to 15 minims three or four times a day (the infusion in 1 to 1½ drachms doses); if the reaction does not begin within four or five days,

the dosage may be increased until symptoms of nausea, diarrhœa, headache or retardation of the pulse appear. It not infrequently happens that the desired fall of heart rate first comes when other signs of intoxication are manifested ; if these persist for several days the drug must be reduced or omitted; whether the rate has fallen or not. The dosage is also reduced if the heart rate falls, and the reduction is continued so long as the heart rate remains below 90. It may be diminished to nothing in many cases ; often 5 minim doses are eventually found to suffice. Usually the full reaction is obtained after six or eight drachms of the tincture or an equivalent quantity of infusion has been given. Whenever the rate has reached 60 or 80 per minute, the drug is stopped, and it is given again only if the heart rate begins to accelerate once more. The appearance of coupled heart beats (Fig. 44) is always a danger sign ; whenever they appear the digitalis must be discarded. I have seen more than one case of unexpected death, attributable to excessive dosage with digitalis, at this stage ; it must be remembered always that digitalis is a poison, and that it has other actions than the simple reduction of heart rate.

In most instances where the patient has reacted, the drug can be stopped without the recurrence of accelerated rate so long as he remains in bed. When he rises from bed a renewal of the small dose (5 minims) may be necessary. In other cases the result is less satisfactory and heavier dosage must continue.

It sometimes happens that a patient is peculiarly intolerant to digitalis, and that, where a reaction is expected, a dosage of 15 to 20 minims of the tincture cannot be reached or maintained sufficiently long, without nausea, or other discomforting symptoms supervening. Strophanthus or squills may be tried, starting with doses of 10 minims of the tinctures. These drugs are pushed in the same manner, but

though they are less apt to induce nausea or vomiting, and while diarrhœa is the chief intestinal disturbance produced by them, they are less reliable than digitalis. In some of these cases, too, recourse may be had to intravenous injections of strophanthin.

When a patient who has fibrillation is first seen, and the heart beats persistently at 170-200 per minute, the condition is urgent and heavy doses of digitalis (minims 20 to 30) should be employed. The intravenous injection of strophanthin is also valuable at such times. Two or three doses of 1/250 of a grain, each in 40-60 minims of saline, are given at an interval of two hours. The reduction of rate is almost immediate, and heart rates of ninety or eighty are reached in from 6-12 hours. The remedy should be employed cautiously and its adoption must be confined to the urgent case which belongs to the rheumatic group or to similar cases in which medication by the mouth has been hindered by the onset of gastro-intestinal symptoms.

A relatively small group of cases of auricular fibrillation remains where, with persistently high ventricular rate, digitalis has little or no influence ; these patients are also unaffected by strophanthus and squills. In so far as the fibrillation and excessive heart rate are concerned, nothing further can be done for them.

The treatment of the case of auricular fibrillation, in the patient who is up and about, is guided mainly by the rate of the heart and the urgency of the patient's symptoms. The disorder is generally persistent, and most hospital patients eventually leave their beds and return to their former occupations. But even where the pulse rate is persistently low and symptoms are few, excessive exertion should be avoided ; heavy manual labour, strenuous games or sports should form no further part in the daily life. If the pulse rate quickens readily, if drugs are constantly required to maintain the

retardation, and especially if breathlessness or precordial uneasiness are easily induced, further restrictions are necessary. All patients of the female sex should be specially warned of the strain and danger of pregnancy.

Regular meals consisting only of a sufficiency of solid and sustaining food, preferably dry; early hours; a placid existence; the avoidance of public buildings and all places and seasons in which influenza and bronchitic troubles are contracted; and, lastly, scrupulous attention to the hygiene of the teeth and throat, are sound directions in this as in other serious heart maladies.

Belladonna, its allie, hyoscyamus and their extractives should be avoided. Their customary action is to increase the rate of the ventricle considerably in this condition.

In cases of urgency, or where the patient's life may be considerably prolonged by surgical operation, general anæsthetics may be employed. But where there is any hesitancy to perform an operation, apart from the cardiac condition, the presence of fibrillation should countermand it.

Paroxysmal fibrillation.

Most hearts which develope fibrillation of the auricles maintain this mechanism to the end of the chapter; it is essentially a chronic and terminal malady. But from time to time transient attacks are seen, and in some patients paroxysms of fibrillation of a few hours, days or weeks duration are noted. The affection, when it takes this form, is generally classed as paroxysmal tachycardia. In my discussion of paroxysmal tachycardia I have excluded it, desiring to deal, as I did, with the simpler mechanism alone.

The exact frequency of the paroxysmal affection has not been ascertained, but it may be gauged approximately by comparison. Of the 126 cases of auricular fibrillation included in the table on page 75, in only 12 was the disorder

temporary and recurring. Paroxysms of regular tachycardia appear to be more common; as opposed to the 12 irregular tachycardias, regular tachycardias have been seen in 29 patients.

The symptoms of paroxysmal fibrillation have been spoken of already. They may be inconspicuous or profound. When the rate of ventricular response is rapid (160-200 per minute) the symptoms are those of simple tachycardias at similar rates, though they are on the whole more severe. The prognosis is reasoned in the manner stated for regular paroxysms; the management and symptomatic treatment of the attacks are similar in the two. A few words are necessary upon digitalis medication. Drugs belonging to this group have been known, not infrequently, to excite fibrillation in those predisposed. They are therefore contra-indicated in paroxysms of short duration and in those which produce few symptoms. Where the paroxysms are more prolonged, or where the symptoms are urgent, they may be given to advantage; and, if given, dosage should be arranged for a speedy reaction. In such patients, the reaction consists of slowing of the ventricular rate and is therefore beneficial, but the duration of the paroxysm is usually prolonged by the administration.

CHAPTER VII.

ALTERNATION OF THE PULSE.

Definition.

A condition in which the left ventricle, while beating regularly, expels larger and smaller quantities of blood at alternate contractions.

The mechanism in alternation of the pulse.

Alternation in the size of pulse beats, so that each alternate beat is large and each alternate beat is small is of obscure origin. The contractions of the ventricle are regular, and each is preceded at a normal interval by a contraction of

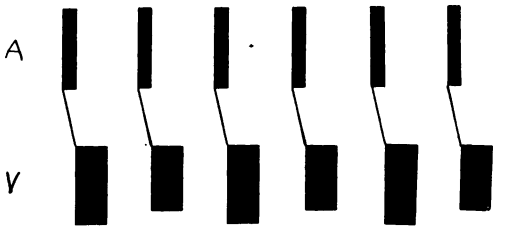

Fig. 45. A diagrammatic representation of alternation of the heart. The auricular and ventricular beats are placed regularly and in order, but alternate ventricular contractions are weak.

the auricle (Fig. 45). The disturbance is dependent upon some unexplained anomaly of the ventricular systoles, whereby at each alternate systole of the left ventricle a greater or lesser quantity of blood is thrown into the systemic

arteries. In the figure, I have represented this anomaly by varying the size of the ventricular rectangles.

Etiological and pathological relations.

Alternation of the pulse is seen in two classes of patient.

First, it occurs in those in whom the heart rate is unduly accelerated and more especially as an accompaniment of paroxysmal tachycardia. Associated with paroxysmal tachycardia, it has etiological and pathological relations in common with the last named disorder; its prognostic significance is not fully known, but, as it depends chiefly, if not entirely, upon acceleration of the heart rate, it may be regarded almost as a physiological reaction to the increased frequency of beat.

Secondly, it occurs when the heart rate lies within normal limits and at such times it is a sign of much clinical value. Seen in elderly subjects and pre-eminently in the male sex, it consorts especially with angina pectoris, high arterial pressure, renal disease, and fibrotic myocarditis. It has been seen in pneumonia during the pre-critical stage, and also in patients under the influence of large doses of digitalis.

It is encountered in experiment under similar circumstances, namely, when the heart rate is extremely rapid, or when it has been injured by the intravascular injection of poisons.

Whenever it occurs, there is reason to believe, either that a tolerably healthy heart muscle is carrying an excessive burden, or that a diseased or poisoned muscle is struggling to perform work of which it is barely capable.

In the remainder of this chapter, I shall allude to pulse alternation as an accompaniment of heart rates which are not high. When the heart is disposed to alternate, the actual alternation is unmasked by anything which imposes a fresh and added strain upon that organ. Thus it is often

made manifest by a slight acceleration of pulse rate ; and, in the earlier stages of its development, it is frequently brought to light by the occurrence of a single premature beat ; under the last named circumstance it follows immediately upon the disturbance, and continues for a varying number of heart cycles.

The recognition of pulsus alternans.

It is an unfortunate fact, but nevertheless true, that most instances of *pulsus alternans* cannot be recognised by other than instrumental means. There are patients in whom it affects the pulse continuously, and in whom alteration in

Fig. 46. Alternation of the pulse. Each alternate beat is strong and each alternate beat is weak.

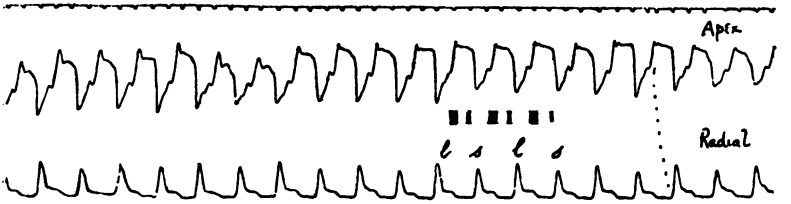

Fig. 47. Apex and radial curves and heart sounds in heart alternation. The curve is taken at a faster rate than the last and shows the slight variation in pulse pauses. As opposed to the picture when premature beats are present, the *stronger* beat is followed by the *longer* pause.

the force of alternate pulse beats is perceptible to the finger* ; but such cases are rare, and identification of the condition from the feel of the pulse is most uncertain. Examination of the apex beat gives little assistance, for the heart beats with regular

* The separation from a dicrotic pulse is easy ; for where the latter is present the beating of the pulse is at twice the rate of the ventricle.

rhythm and the differences in the force of ventricular systoles and the intensity of the heart sounds are inappreciable.

It is a sign of such importance and is so readily overlooked, that it should be sought deliberately whenever there is reason to suspect the possibility of its presence. Thus it is wise to examine all cases of angina pectoris, all cases of high blood pressure and all elderly subjects in whom affection of the heart is suspected, or renal disease is known to exist, with a specific object, namely, to determine its presence or absence. It should be looked for, too, in all elderly people in whom premature beats are frequent. If such methods are adopted it will not often escape detection. It is so frequently confined to the few cycles which follow a premature beat that, in any one class of patients mentioned, it is most useful to obtain a curve which contains such a beat. This may often happen at the first examination. The patient should remain standing, for premature beats are more frequent in this posture, and if he has come some distance, it is well that the examination should proceed at once, since premature beats are more conspicuous at such times. It should be remembered too, that a held breath may evoke a premature beat and the opportunity of catching it in this manner should not be lost.

Fig. 48. Alternation of the pulse, appearing after, and as a result of, a single premature beat *p*. It lasts for four heart cycles.

Single premature beats are usually followed by a pulsation of exceptional size, for the heart puts out more than its usual quantum of blood. It is the pulse which succeeds this tall beat which shows the first sign of alternation; it is less forcible than that which succeeds it. In Fig. 48 a regular pulse is

interrupted by a single premature contraction (p); it is followed by the usual pause and this is succeeded by a tall pulsation (l); the next beat s^1 is small, it is followed by a taller beat l^1. The small beat s^1 is, as I have said, the earliest sign of the condition, and it may be the sole sign. In the actual figure s^2, the alternate beat, is also low. Alternation has proceeded for four cycles before the normal pulse beats are restored. In Fig. 46 and 47 the condition is persistent throughout each curve; little and big beats are arranged alternately. Extreme degrees of alternation of the pulse are seldom encountered; but on very rare occasions the little beats vanish entirely, and the pulse rate is halved.

The other irregularity with which *pulsus alternans* may be confused is a coupled pulse resulting from premature beats, but this only happens where the prematurity of the second beat of each couple is slight. An example is shown in Fig. 33 of an earlier chapter. There is a sufficient contrast between them; whereas the little beat in Fig. 33 is followed by the longer pause, if *pulsus alternans* is present the little beat is followed, if there is any variation in pauses, by a slightly shorter pause. In tracings written upon slowly travelling paper the difference in intervals is hardly perceptible (Fig. 46); but where the paper has moved faster, a measurable difference is often found; it is well seen in Fig. 48, in which the pauses following l and l^1 are longer than those following s^1 and s^2.

The subjective sensations of patients presenting pulsus alternans.

Alternation of the heart gives rise to no recognised symptoms; the patient complains of sensations which are referable to other causes. Thus, anginal pain is common. Breathlessness is even commoner; it is often nocturnal, repeatedly awakening the subject of it after short periods

of sleep and being accompanied by acute anxiety. Breathing of the Cheyne-Stokes type is rarely noted by those who manifest this respiratory abnormality, but periodic dyspnoea may be remarked by the friends, especially by those who sleep with the patients.

The prognosis.

Alternation of the pulse belongs to a small group of phenomena witnessed by those who attend the sick, which, treated as isolated signals, are in themselves emphatic and portentous. It ranks with *subsultus tendinum*, with *optic neuritis*, with the *risor sardonicus* and other ill-omened messengers. It is the faint cry of an anguished and fast failing muscle, which, when it comes, all should strain to hear, for it is not long repeated. A few months, a few years at most, and the end comes.

How grave is the condition of the patient whose heart produces this alternating pulse is often witnessed to by associated signs ; angina, nocturnal dyspnoea, Cheyne-Stokes breathing or high blood pressure are often encountered in the same subject. But here lies its special significance : each and everyone of these signs may fail, and alternation may appear alone to foretell the future. Unexpected death is a common termination.

I write of continued alternation, of the pulse which alternates in force for many cycles. It is persistent while the heart yet lives. The prognostic value of the lesser grades of perverted mechanism are less certainly known ; but that their significance is grave, and that they are but too often the forerunners of the fully developed condition, is understood. A favourable prognosis is always forbidden by the last, and can be but rarely justified in the presence of the first. The only propitious circumstances are a history of exceptional and prolonged strain in the patient who shows the sign, strain which may be at once and permanently avoided, and evidences of acute intoxication which is vanishing.

The treatment.

The management of heart cases, in so far as it is affected by the presence of alternation, may be stated in a few words, for it should be evident. Alternation is a sign of overtaxation; it demands relief. In the busy it calls for prompt and drastic curtailment of the work, be it mental or physical exertion. In the more sedentary, it is an indication for prolongation of the hours of actual rest, both of body and mind; the condition of such patients may undergo temporary relief by a long period of absolute quiescence. In each case the avoidance of all sources of anxiety or emotion is to be enjoined. The presence of alternation forbids the administration of general anæsthetics in major operations, unless the withholdment of the first immediately jeopardises life, or unless one or other is necessitated for the relief of intolerable pain.

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THE PETER BENT BRIGHAM HOSPITAL
BOSTON, MASSACHUSETTS

27. Oct 1914

Dear D. Jackson,

It is most kind of you to ask me to dinner on Friday & I shall be most pleased to come

Yrs sincy

Thomas Lewis